PRINCIPLES OF PLANT PATHOLOGY

A Text and Laboratory Manual

PART II

OWENS

PAMPHLET COLL.

Principles of Plant Pathology

LABORATORY MANUAL

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Part II

BY

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AT

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PREFACE

The subject-matter presented in this manual has been selected to meet the needs of students in agriculture for a general course dealing with plant diseases and their control.

Plant pathologists have long been aware, and practical agriculturists should fully realize, that one of the important factors which interfere with maximum crop production as well as lower the quality of the product is the prevalence of plant diseases. When this fact is fully comprehended the conclusion necessarily follows that, in order to attain maximum production and to insure a product of high quality, diseases must be controlled.

In this volume the fundamental principles underlying the cause and control of plant disease will be emphasized. However, in order that these laws may be more thoroughly understood, some of the more important plant diseases of various types are outlined for study in such manner as to illustrate the applications of the principles involved.

Accordingly the treatment of the subject naturally falls into two parts as follows:

Part I.- The first part of the work consists of a discussion of topics of general interest concerning the nature, cause and control of plant diseases. The subject-matter of these general topics is indicated by the chapter headings, as, for example, historical account of the rise and development of the science of plant pathology, definition of plant disease, symptoms and signs of disease, classification of plant diseases, relation of plant diseases to environment, methods of investigating plant diseases by means of pure cultures and inoculations, methods of control such as the use of fungicides, quarantine and inspection, clean seed and nursery stock, immunization, storage and transportation problems, sick soil, relation of insects to plant diseases, and unsolved problems of plant pathology.

Part II.- In this part will be found a series of exercises covering representative types of plant diseases. The diseases outlined for laboratory study have been selected from as wide a field as possible, both as to causal agent and as to host, in order to give the student as broad a conception of the whole field of plant pathology as is possible for him to get in the limited time devoted to a course of this kind. Attention is called to the fact that under each disease listed the exercise falls into three subdivisions, namely, (1) directions for laboratory work, (2) a text discussion of the disease, and (3) a selected list of references to original literature on the subject.

For a number of years the writer has tried to present this course by sending the students to the library to look up the sources of information for themselves. This was found to be impractical with large classes of undergraduates because it required more of the student's time to get the desired material together than was available in his schedule for this course. Therefore, an attempt has been made to bring at least a part of the desired information together in this form. The

compilation has been accomplished in large part by making extensive quotations from original sources, such as bulletins and periodicals, and from books, wherever authentic and recent information could be found, on both the general subjects discussed in Part I and on the specific diseases outlined in Part II. Care has been exercised to give proper credit for all quoted matter, the quotation being inclosed in quotation marks and accompanied by the reference citation. It is felt that the compilation of essential facts under one cover as here presented will be a great time-saver for the student in thus making available for his study a selected list of the best articles on the subject without requiring so much of his time in looking up references in the library.

Acknowledgement is due H. P. Barss, Professor and Head of the Department of Botany and Plant Pathology at Oregon Agricultural College, whose interest in the work and whose helpful suggestions and criticisms have been of great value to the writer in compiling this manual. The writer has also consulted freely with Professor M. B. McKay and Dr. S. M. Zeller, Pathologists of the Oregon Experiment Station, upon subjects with which they are especially familiar.

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Corvallis, Oregon,
September, 1924.

CHAPTER XV.

General statement of laboratory problems.

As stated in the preface, the second part of this manual is devoted to the careful study of a selected list of plant diseases. This list has been chosen to illustrate the different types of plant maladies as discussed in Part I under classification of diseases. (See Chapter III).

Since it is assumed that any student who pursues this course must first have had a general course in botany, but more especially since, in order to apply control measures intelligently, it is absolutely necessary to know the cause of the disease, it has been deemed best to classify the diseases outlined in this manual on the basis of the causal agent of the disease. (See under point F on page 24).

Method of study.

In order that the student may have in mind a definite plan or model to follow in taking up the study of any specific disease the following tentative outline is offered. It is not intended that this outline shall be followed rigidly in all cases, but it will serve as a guide and can be modified to fit the needs of any particular case.

1. Laboratory work.- The work in the laboratory should include the following points:

a. Symptoms.- The symptoms of the disease should be carefully observed and full notes taken. The student should constantly keep in mind the purpose in this study of symptoms; namely, to acquire the ability to identify the disease wherever it may be seen.

b. Pathological anatomy and histology.- Wherever possible, sections of the diseased parts should be studied under the microscope, in order to determine the effect of the parasite upon the tissues and cells of the host.

c. Morphology and life history of the causal organism.- This should include a detailed microscopic study of both the vegetative and reproductive phases of the organism. A thorough knowledge of these phenomena is absolutely necessary in order that one may have a rational working basis for the control of any plant disease.

d. Culture work.- If time and equipment permit it will be of interest to the student to make isolations and pure cultures of some of the disease producing parasites. Detailed instructions for carrying on culture work may be found in the following texts:

Duggar, B. M.- Fungous Diseases of Plants. pp. 9-40.
Smith, E. F. - Bacterial Diseases of Plants. pp. 76-131.
(See also Chapter IV of this manual)

e. Drawings and notes.- A few careful drawings should be made to illustrate the chief features observed in the laboratory study of the diseased specimens. When the drawings are finished, complete notes should be written including a description of all points observed in the laboratory. These notes should be supplemented with additional points obtained by reading the references cited at the end of each exercise.

2. Lecture and recitation.- The class work aside from the laboratory shall consist of lecture and recitation interspersed. The lecture shall cover topics of general interest concerning the nature and control of plant diseases. The recitation shall consist of oral or written quiz over the work previously presented in the laboratory and lecture and over assigned reading.

3. Field Work.- Whenever it is practicable to take up any field work, any or all of the following topics may occupy the attention of the class:

a. Identification.- It is desirable that the student be able to identify, in all its forms, any disease studied, whenever he sees it in the field.

b. Observation of development.- If possible, students should make a series of observations, extending over a considerable length of time, in order to note different stages in the development of any particular disease.

c. Estimation of losses.- When a disease has reached the stage at which fairly accurate estimates can be made, the student should estimate percentages of loss. This will help to impress upon his mind the seriousness of plant diseases and the necessity of working out practical control methods.

4. Reading.- As each disease is studied it is desirable that the student read some of the best articles that have been published on this disease. The reading should be done before the notes on the exercise are completed, in order that important facts recorded in the literature but not observed by the student in his laboratory study may be included in the notes of the completed exercise. Some of the more important sources of information on plant diseases are listed below.

a. Books.

(1) Whetzel, H. H.- History of Phytopathology. 1918.
(2) Duggar, B. M.- Fungous diseases of plants. 1909.
(3) Stevens and Hall.- Diseases of economic plants. 1910.
(4) Stevens, F. L.- Diseases of economic plants. (revised edition). 1921.
(5) Stevens, F. L.- The fungi which cause plant disease. 1913.
(6) Hesler, L. R. and H. H. Whetzel - Manual of fruit diseases. 1917.
(7) Smith, E. F.- Bacteria in relation to plant diseases - 3 volumes 1905.
(8) Smith, E. F.- Bacterial diseases of plants. 1920.
(9) Rankin, W. H.- Manual of tree diseases. 1918.

(10) Taubenhaus, J. J.- Diseases of truck crops. 1918.
(11) Taubenhaus, J. J.- Diseases of greenhouse crops. 1920.
(12) Freeman, E. M.- Minnesota plant diseases.
(13) Cook, M. T.- Diseases of tropical plants.
(14) Harshberger, John W.- Mycology and plant pathology. 1917.
(15) McAlpine, D.- The smuts of Australia. 1910.
(16) McAlpine, D.- The rusts of Australia. 1906.
(17) Massee, Geo.- Diseases of cultivated plants and trees. 1910.
(18) Hartig, Robert.- Diseases of trees. 1894.
(19) Tubeuf and Smith.- Diseases of plants. 1897.
(20) Sorauer, Paul.- Handbuch der Pflanzenkrankheiten. English Translation by Dorrance. 1922.
(21) Fryer, P. J. Insect Pests and Fungous Diseases. 1920.

b. Journals and Periodicals.
(1) Phytopathology.
(2) Journal of Agricultural Research.
(3) Annals of the Missouri Botanical Garden.
(4) Botanical Gazette.
(5) Mycologia.
(6) American Journal of Botany.
(7) Annals of Applied Biology.

c. Popular Periodicals.
(1) Better Fruit.
(2) Oregon Grower.
(3) Pacific Homestead.
(4) Oregon Countryman.
(5) Monthly Bulletin of the Department of Agriculture, State of California.
(6) American Fruit Grower Magazine.

d. Bulletins, circulars and reports.
(1) Of the various state experiment stations.
(2) Of the extension divisions of state agricultural colleges.
(3) Of the United States Department of Agriculture.

e. Extensive bibliographies and lists of literature on plant diseases may be found in the following:
(1) Agricultural Index.
(2) Botanical Abstracts.
(3) Experiment Station Record.

5. Notes and reports.- The following suggestive outline will be found useful, either in writing up the notes at the end of a laboratory period, or in writing a more extended report upon any disease. This outline cannot be followed strictly in all cases but can be easily adapted to any particular disease. It is recommended that the student follow it as closely as possible in writing the notes.

Name of Disease.

Caused by (Insert scientific name of causal organism).

1. Historical account.

2. Present geographical distribution.

3. Losses due to this disease.

4. Hosts.

    a. All species and varieties attacked.
    b. Relative susceptibility.

5. Symptoms.

6. Morphology and life history of causal organism.

    a. Vegetative phase.
    b. Reproductive phase.
        (1) Perpetuation
            (a) Spore forms.
                (1) Conidial stage.
                (2) Perfect stage
                (3) Other spore forms.
            (b) Sclerotia.
            (c) Vegetative mycelium.
            (d) Simple cell division (as in bacteria).
        (2) Dissemination
            (a) Time
            (b) Agencies
        (3) Infection
            (a) Time
            (b) Place
            (c) Manner of
            (d) Environmental factors.
                (1) Favorable
                (2) Unfavorable

7. Control measures.

8. List of references.

CHAPTER XVI.

Diseases caused by slime molds.

The slime moulds constitute a group of plants occupying a position very low in the scale of plant life. They were formerly classed by some authors as a subgroup of the fungi but are now usually considered as a distinct group of plants coordinate with the fungi.

The character which distinguishes this group from others is the fact that the vegetative phase consists, either of distinct amoeboid cells, or of a mass of naked protoplasm, the plasmodium, which in turn is composed of numerous cell units without cell walls. The amoeboid forms and the creeping plasmodium have led to doubt as to whether the slime molds are really plants or animals. They are usually considered plants, however, because in the fruiting stage they produce walled spores.

The great majority of the slime moulds are saprophytic and live on decayed wood, leaves and other vegetation. Such forms are usually of no economic importance, although occasionally a saprophytic form may grow up over a living plant, exerting a smothering effect upon it.

On the other hand a very few species of this group of plants are truly parasitic and are responsible for some serious diseases of agricultural plants. It is these parasitic forms in which the student of plant pathology is chiefly interested.

There are only two serious plant diseases caused by slime moulds; namely, Club Root of Cruciferous plants and Powdery Scab of potatoes. Outlines for the study of these two diseases follow.

Anyone who is especially interested in the slime moulds as a group should read the book "North American Slime Moulds" by McBride.

CLUB ROOT OF CABBAGE

Caused by Plasmodiophora brassicae Wor.

This disease is one of the few caused by parasitic slime moulds. It is a serious disease of the roots of cabbage, turnips and other members of the mustard family.

1. Laboratory study.

a. Symptoms.- Examine specimens of diseased plants, photographs and other available illustrations, and write a detailed description of the symptoms as observed. Note the peculiarities of the enlargements on the roots. Some of the

common names which have been applied to this disease, such as club root, fingers and toes, maladie digitoire, are suggestive of the characteristic symptoms. Draw a typical diseased specimen.

b. Effect upon the structure of the host.- In what part of the plant does the enlargement occur? Are all of the roots involved? Does the overgrowth involve the entire diameter of the root or is it simply an outgrowth from one side? Examine sections of the diseased root under the microscope. Are all of the tissues involved or is the disease confined to certain tissues? Is the enlargement of the root due to multiplication of cells (hyperplasia) or to enlargement of individual cells (hypertrophy) in the root? See the text, Fungous diseases of plants, by B. M. Duggar, page 100; also Vermont Experiment Station Bulletin 175, page 5. Make a drawing from the section to illustrate these features.

c. Morphology and life history of causal organism.

(1) Vegetative phase.- This consists of amoebae or plasmodia, that is, naked masses of protoplasm which live within the cells of the host. Examine sections of diseased root under the microscope and find cells filled with the protoplasm of the parasite. These cells can be distinguished by their denser and more granular contents. Draw. See also the following references: Duggar, B. M., Fungous diseases of plants, pages 99-100. Cornell Experiment Station Bulletin 387, page 434. Vermont Experiment Station Bulletin 175, pages 5 and 16.

(2) Reproductive phase.- Reproduction of the club root organism is effected by means of spores which are formed by the breaking up of the protoplasm of the slime mould into small fragments and the formation of cell walls around these bits. A large number of spores may be formed from the plasmodium within a single cell of the host. Find such spore-containing cells in the section and draw. See illustrations of this feature in the references cited above.

(3) Perpetuation, dissemination and infection.- Consult the references and find out how the spores are disseminated, their manner of germination, the place and manner of penetration of the host, and the manner of spreading within the host.

d. Notes.- Write up a complete discussion of this disease, including points observed in the laboratory as well as additional details obtained from a reading of the references. For a suggestive model in writing up the notes, see the outline on page 130.

2. Recitation.- Before the class meets for the recitation, read some of the references and quotations in the following list and be prepared to discuss all details brought out in these articles, especially the parts dealing with life history and control measures.

Text discussion.

The distribution, symptoms and certain phases of the life history of this disease are discussed briefly by Duggar (1) in the following paragraphs:

"The club root, or club foot, is an unsightly and destructive root disease of crucifers which has been known in Europe for considerably more than a century. In England it is commonly called fingers and toes, anbury, etc. (Germany, Kohlhernie: France, maladie digitoire). Our knowledge of the casual relations of a Myxomycete, Plasmodiophora, to the disease is primarily based upon the excellent researches of Woronin published in 1878.

"Habitat relations. In Europe the fungus is quite generally distributed throughout the market-gardening sections. In 1876 Woronin estimated the losses due to it in the vicinity of St. Petersburg at $225,000. In the United States it has been disastrous in many of the northeastern states, particularly in those trucking regions which supply the markets of New York and Boston. It is, however, occasionally found both South and West. The limits of its distribution have not been clearly defined. Unquestionably it thrives best in a rich, warm, moisture-retaining soil.

"Seedling plants affected by this parasite show a decided "flagging". They are stunted, unhealthy in appearance, and they may gradually die. Few of those affected when young reach maturity. The parasite attacks the roots and gains entrance to the parenchymatous tissues. The presence of the organism within the cells affords a stimulus to abnormal growth. There results, in fact, malformities of striking appearance. These vary, on the one hand, from slight nodose swellings in the small rootlets, and knotty masses in the tough roots of some weeds, to the more or less irregular, but generally fusiform, digitate swellings in the cabbage, and the lobulated enlargements of the turnip.

"Many members of the mustard family, Cruciferae, are subject to the attacks of this fungus. A complete list of the hosts upon which it has been found cannot be given on account of the fact that much information has been covered up by too general statements. In the United States, however, it certainly occurs upon varieties of cabbage, cauliflower and Brussels sprouts (Brassica oleracea), turnip (Brassica campestris), rutabaga (Brassica rapa), radishes (Raphanus sativa), and certain mustards (Sinapis and Brassica). It has also been found upon such weeds as shepherd's purse (Capsella Bursa-pastoris) and hedge mustard (Sisymbrium officinale).

"Morphology.- A microscopic study of sections of the diseased root shows that the organism is most abundant in parenchymatous cells, often in the vicinity of the cambium. There is in quantity an abnormal development of phloem. The xylem portions of affected roots are relatively inconspicuous. According to some observers, certain bundle elements may also show the parasite.....

"In an earlier stage the contents of the infected cells is of half-fluid consistency, later turbid, and finally granular. Even in the first stage the parasite is noticeable in the amoeboidal form and the nuclei may be distinct. The number of amoebae is increased by division, probably by a kind of budding process. Starch is present in the host cells but the amoeba gives only a reaction for oil. Several nuclei are present in each amoeba, and as the number of the latter is increased they become rounded and pressed closely together into what is practically a plasmodium. The spore-forming stage is then initiated. There may be successive simultaneous divisions, and then the spores are differentiated by the formation of a cell wall around each nucleus and surrounding cytoplasm.

"At maturity most of the pathological cells are packed full of the spherical thick-walled spores, and the latter are perhaps set free only by the disintegration of the roots. Certain unusual appearances, moreover, have been described, but these are not understood. In from four to twenty-four hours the spores will germinate in water in which some of the host tissue has been teased out, the contents of each spore escaping in the form of an irregular protoplasmic mass which may quickly change its form. There is at first, for the most part, an appearance of an elongated process or cilium, which doubtless permits rapid motility, denoting also a swarmspore stage. In the swarmspore stage a nucleus and a pulsating vacuole are always seen. Later, the protoplasmic mass moves wholly by amoeboidal streaming."- Duggar. Fungous Diseases of Plants. pp. 100-102.

Perpetuation.- The organism is perpetuated by means of the resting-spores. These remain in the diseased roots until they are liberated by the decay or disintegration of the roots when they are released into the soil.

Dissemination.- The disease may be disseminated in various ways. The transfer of diseased soil on farm implements, wagon wheels and on the feet of animals and man is entirely possible. It may be carried wherever diseased plants or parts of plants are transported. Seedlings transplanted from a diseased seed bed are almost certain to carry this disease with them.

Chupp (3) calls attention to the fact that, while the swarm spores are more or less motile, they can travel only very short distances in the soil water under their own power and that this method of dissemination is negligible. He also states that it is doubtful whether the wind is a very potent factor in disseminating the spores since this would be possible only in loose, dry soils and during high winds.

Infection.- Chupp (3) states that his experimental work showed that infection takes place through the basal portion of the root hairs. He believes that direct penetration into simple epidermal cells seldom, if ever, takes place.

In a later work on this subject Kunkel (4) demonstrated that infection can take place through epidermal cells, other than root hairs, even on the stems of quite large plants and on the older roots.

Distribution of the parasite within the host.- It is generally agreed that after a swarm spore, or amoeba enters the host cell it migrates to other cells by direct cell wall penetration. It may also be distributed to new cells by cell divisions. When a host cell containing an amoeba divides the parasite is also divided, a part going to each daughter cell. Kunkel (4) claims that most of the spreading through the tissues is accomplished by the migration of amoebae through the cell walls and that host cell divisions play a minor part in this distribution.

Histology of the club.- Kunkel (4) gives a very good description of the manner in which the tissues of an invaded plant are modified. The essential facts as stated by him follow:

"Like other dicotyledonous plants, the cabbage during its secondary growth produces, on either side of the cambium, cells that sooner or later become

differentiated into xylem and phloem. In the case of the cabbage plant, the differentiation takes place more quickly on the xylem than on the phloem side. The development of xylem tissue thus keeps pace with the increase in transpiration, due to the growth of leaves. When the young root or stem is attacked by the parasite in question the cambium quickly becomes infected, as has been seen. The plasmodia then pass into the layers of undifferentiated cells on either side of the cambium. These cells, instead of developing into vascular elements, as they do in healthy plants, are stimulated to abnormal growth and division. Even the non-infected cells surrounding those that contain the parasite are to a certain degree prevented from developing into the vascular tissue. The differentiation of non-infected cells is, however, not entirely prevented, and a small amount of vascular tissue continues to be added to each bundle. The top of the diseased plant keeps on growing--not so fast as in the case of healthy plants--but much too fast for the atrophied development that takes place in the conducting system. In order words, the leaf surface outgrows the conducting system. The parasite may not interfere with the functioning of this system, but it prevents that enlargement which would be necessary to meet the needs of the ever-increasing number of transpiring cells. The cells of the infected tissues, instead of contributing to water conduction, use up water in their growth. Furthermore, the large swellings themselves, especially when above ground, increase the transpiring surface. Finally on a warm, dry day the critical point is reached. The leaves are no longer able to obtain as much water as they transpire and the plant wilts or, as the gardeners say, 'flags'.

"But not all plants are attacked while still young. It often happens that they escape infection in the seed bed, and contract the disease only after having reached a considerable size. These plants have well-developed vascular systems before they become infected. Can the parasite cause wilting in such plants and, if so, by what means?

"In the study of the infection of old stems we have seen that the plasmodia are able to attack the undifferentiated tissues on either side of the cambium, but that it can not penetrate far into the older portions of the bundles. It is unable to attack the woody cells of the xylem, but the undifferentiated cells of the medullary rays are still susceptible. The invader is able to penetrate the rays and to stimulate their cells, so that instead of remaining small and inactive they grow and divide and give rise to a pathological tissue.....

"The growth of the medullary ray cells splits open the woody cylinder. The xylem tissues are forced apart and the bundles distorted in a variety of different ways. Sometimes the splitting up of these tissues is so complete that no two vascular strands can be found near together. In other cases the invasion of the medullary rays is not so complete, and the splitting of the woody cylinder is only partial. It frequently happens that the wood is split into two approximately equal halves."- Kunkel, L. O. Tissue Invasion by Plasmodiophora Brassicae. Journal of Agricultural Research 14: 558-559. September 16, 1918.

Control.

Cunningham (5) gives the following summary of recommended and suggested control measures:

"Observations on the susceptibility of crucifers to club-root indicates that the great majority of, if not all, crucifers are susceptible. A few varieties were not infected on our plots. The range varies all the way from 100 per cent susceptible to 100 per cent immune.

"Cabbages show variety resistance to the attack of club-root as revealed: (1) by the percentage clubbed, (2) by the number of saleable heads, and (3) by the weight of saleable heads per acre. Among the more susceptible varieties of cabbages are Mammoth Rock Red, Dark Red Erfurt, American Savoy, Perfection Savoy, All Seasons and Volga, while among the more resistant varieties may be included Hollander, Stone Mason, Large Late Flat Dutch and Henderson's Early Summer. No variety escaped, indeed none showed more than 26 per cent of healthy plants. Mammoth Rock Red and Perfection Savoy were the worst clubbed, and Hollander the least clubbed.

"Radishes show a wide range of susceptibility to club-root, varying from the Long Scarlet Radish (92.2 per cent clubbed) to the Giant Stuttgart (5.6 percent clubbed). No variety was completely free from infection. The disease appears on the radish in addition to the common form as dark discolored spots and fissures.

"The range of susceptibility among the turnips (Brassica Rapa) and rutabagas (Brassica campestris) is even greater than among the cabbage or radishes, varying from Southern Curled Turnip (100 percent clubbed) to White Swede, Sweet Russian, and Sweet German free from Clubbing. Many varieties are fairly resistant. The soft, white-fleshed turnips seem to suffer more than the firm, yellow-fleshed rutabagas.

"The wide range of susceptibility among the species and varieties offers a hope that among the cabbages, turnips, radishes and the more generally cultivated crucifers there may be some varieties which will prove still more resistant to the parasite than are the ones commonly cultivated.....

"The application of lime, preferably air-slaked lime, at the rate of 150 bushels per acre induced a yield of 23,082 pounds of cabbage as compared with one of 672 pounds per acre where no lime was applied; and an almost equally pronounced result was attained the next year on the same plots. Lime to be effective must be thoroughly worked into the soil to a depth of 6 to 9 inches or more, as it does not otherwise readily penetrate the soil.

"Hilling-up cabbage stalks caused the formation of adventitious roots above the diseased roots and materially increased the crop in 1912. No such beneficial results were attained in 1913, indeed, damage was done, possibly because of the lack of rain. The 1912 yield on some hilled plots was increased more than ten-fold. Neither excessive hilling nor hilling during drought is advisable."- Cunningham, G. C. Studies of Club-root II. Disease resistance of Crucifers; methods of Combating Club-Root. Vermont Experiment Station Bulletin 185, November, 1914

Crop rotation.- In case of any disease where the soil becomes contaminated with the organism, as with the spores of the club root organism, crop rotation should be practiced in order to let the soil have an opportunity to become free of the organism. Some crop which is not susceptible to the disease should be grown on the land for a few years.

Sanitation.- Diseased plants or the roots of diseased plants should not be left to decay on the ground and thus contaminate the soil. Seed beds should be kept free of all weeds of the mustard family and if the soil should become contaminated it should be replaced with clean soil.

Review.

Describe the symptoms of club root. Give details of life history. How does the disease-producing organism gain entrance to the host? How does it spread within the host after having gained an entrance? How are the spores disseminated? What do these details of life history have to do with the control of the disease? Suggest control measures with reasons.

References.

(1) Duggar, B. M. Fungous diseases of plants. pp. 97-102.

(2) Lutman, B. F. Studies on club root. I. The relation of Plasmodiophora brassicae to its host and the structure and growth of its protoplasm. Vermont Experiment Station Bulletin, 175. October 1913.

(3) Chupp, Charles. Studies on club root of cruciferous plants. Cornell University Agricultural Experiment Station Bulletin 387. March, 1917.

(4) Kunkel, L. O. Tissue invasion by Plasmodiophora brassicae. Jour. Agricultural Research 14: 543. September 16, 1918.

(5) Cunningham, G.C. Studies on club-root. II. Disease resistance of crucifers; methods of combating club root. Vermont Experiment Station, Bulletin 185. November, 1914.

POWDERY SCAB OF POTATO

Caused by Spongospora subterranea (Wallr.) Johnson.

Powdery Scab is a potato disease which has long been known, under various names, in Europe and was introduced into North America some time prior to 1913, since it was reported from both Canada and Maine in that year.

1. Laboratory study

a. Symptoms.- Make a careful examination of the specimens available and describe the symptoms in detail. See illustrations in Maine Experiment Station Bulletin 227, figures 44-52, and also in U. S. Department of Agriculture Bulletin 82, 1914, plates II and III. Note the different types of symptoms described and illustrated in these bulletins. Note also the distinction between this disease and other diseases mentioned in the Maine bulletin. Make drawings to show typical symptoms.

b. Effect upon the structure of the host.- Lesions may occur on any underground part of the plant; namely, tubers, roots and stolons, but are commonly found on the tubers. Examine sections through diseased regions, also sections through corresponding regions of healthy tubers. Through what layers of cells does the disease penetrate? What is the effect upon these cells? Draw. See Jour. Agricultural Research 4: 270-271 and plates XXXIX to XLIII. See also Maine Experiment Station Bulletin 227, page 93.

c. Morphology and life history of causal organism.

(1) The vegetative phase of the parasite thrives within the cells of the host and consists at first of amoebae which later fuse to form a plasmodium. See illustrations in U. S. Department of Agriculture Bulletin 82, plate I.

(2) The reproductive structures are spores which are formed from the plasmodia within the host tissues. These spores adhere in sponge-like balls. Find spore-balls in the sections and draw. Also see illustrations previously mentioned.

(3) Perpetuation, dissemination and infection.- Look up these topics in Journal Agricultural Research 4: 265-272, and in U. S. Department of Agriculture Bulletin 82, page 11. Note especially the manner of infection as described in the first reference above. Compare this with the manner of invasion in club root of cabbage as described in the fourth reference at the end of the previous exercise.

d. Write complete notes on all of the above points, including information gained both from laboratory observations and from reading the appended references.

2. Recitation.- Before the next recitation period read as many of the following references as possible and be ready to give a detailed discussion of the powdery scab disease of potatoes.

Text discussion.

A few salient facts concerning the history and distribution of Powdery Scab are given by Melhus (3) in the following paragraphs:

"Geographical Distribution of Powdery Scab.- Spongospora seems to be quite generally distributed in northern Europe. As early as 1841 it was recorded as existing in Germany.

"In the Spring of 1913 the Bureau of Plant Industry purchased 22 different varieties of seed potatoes from two dealers in Germany. When these were received and examined by the inspecting pathologist, four of the lots were condemned, being infected with Spongospora.

"In 1846 powdery scab was discovered in England by Berkeley.....

"In the spring of the current year the United States Department of Agriculture imported 18 different varieties of potatoes from Scotland for seed purposes, all of which were found to be infected with Spongospora and were condemned by the inspecting pathologist. Nine different varieties were imported from England for similar purposes and were not allowed to pass, owing to Spongospora infection.

"On October 31, 1913, Mr. W. W. Gilbert, of the Bureau of Plant Industry, collected specimens of powdery scab on potatoes imported into this country from the Netherlands. The following day specimens were taken by Mr. O. A. Pratt and the writer from a shipment coming from Belgium. More recently the disease has been found several times in considerable quantity on potatoes coming from the Netherlands and Belgium. As already stated, the disease has been in Norway since 1886 and has since been found in Sweden.

"It is also interesting in this connection to note that Spongospora occurs in South America, probably the native habitat of the potato.....

"Presence of powdery scab in Canada.- More recently powdery scab has gained a foothold in North America, and early in the spring of 1913 it was reported in several Provinces of Canada by Gussow. .....

"Powdery scab in the United States.- That Spongospora exists also in the United States has been definitely established. In the spring of 1913 Morse, of the Maine Agricultural Experiment Station, obtained some evidence that the disease exists in Nebraska and Massachusetts. No further cases have been reported from these States. In June, 1913, the writer collected 84 tubers infected with Spongospora from four barrels of the Green Mountain variety purchased for experimental purposes at Presque Isle, Me. These had been grown in the vicinity of the village during the season of 1912.

"Spongospora was collected at Washburn, Me., on February 9, 1914, and at Frenchville on the following day. Later it was found at stations farther south in Aroostook County."- Melhus, I. E. U. S. Department of Agriculture Bulletin 82. April 6, 1914. pp. 5-6.

Life history.

Morse (2) gives the following brief discussion of the life history of the powdery scab organism:

"The organism, Spongospora subterranea (Wallr.) Johnson, which is responsible for the production of powdery scab is quite different from the one which causes the ordinary type of scab with which potato growers have been acquainted for many

years. Powdery scab is produced by one of the slime molds which latter belong to the lowest order of living organisms, on the boundary line between the animal and vegetable kingdoms. It is the same type of organism as that which causes the well known club root of cabbage and allied plants. The facts we know regarding its life history may be briefly stated as follows: The earliest stage observed consists of a simple protoplasmic body of the amoeba type located within the young potato cells. The nucleus of this amoeba divides; and generally in the early stages of the infection this is followed by a division of the amoeba itself, which process continues until a number occur in one cell.....

"After the contents of the whole cell are exhausted the amoebae fuse together into a larger body called the plasmodium. This is the first stage of spore formation. Following this, certain other changes take place until finally the plasmodium is converted into a loose, spongy, yellowish or brownish body usually spherical to ovoid in shape and varying in size but averaging about 1-500 of an inch in diameter. These are the so-called "spore balls" of the organism. They are made up of aggregates of many small, spherical spores somewhat less than 1-5000 of an inch in diameter and it is the masses of these spore balls which constitute the powdery substance found in the pustules on the diseased potatoes." Morse, W. J. Powdery Scab of Potatoes. Maine Agricultural Experiment Station Bulletin 227. pp. 92-93. 1914.

Kunkel (4) contributes the following additional information on the life history of the powdery scab organism.

"A Contribution to the Life History of Spongospora Subterranea. The manner of infection of tubers of the potato (Solanum tuberosum) by Spongospora subterranea seems never to have been observed.....This paper deals primarily with certain fundamental facts relating to the life history of the organism and its parasitic relations to the potato, which, as indicated above, are very imperfectly understood. In the author's studies of the germination of the spore balls of S. subterranea it has been found that a large number of small plasmodia are produced by the fusion of the amoebae on certain cultural media. These results, coupled with previous observations in microtome sections of very young sori, show unmistakably that infection takes place through invasion by a plasmodium.....

"In stained sections it is possible to observe in detail the manner in which the potato tissue is attacked by the plasmodium. Some of the preparations show it passing down through and between the epidermal cells. Usually a considerable number of cells are killed at the point where the plasmodium enters. Once beneath the epidermis, it spreads out in all directions and forms a rather flat, disk-shaped mass, which separates the epidermis from the tissue beneath. In general, the potato cells in contact with the plasmodium are at once stimulated to abnormal growth and division, but some of them are killed as the plasmodium spreads out over the healthy tissue. In this way it comes to occupy a space between the uplifted epidermis and sound tissue beneath. This circular plasmodial mass is thicker at the point where it enters the epidermis than toward the edges. At first the lower surface of the disk-shaped mass is almost smooth. It comes in intimate contact with the cells on which it rests. Soon, however, a number of projections of pseudopodia begin to extend downward, push in between the cells of the sound tissue, and seem to crowd them apart. All of the cell walls that have been or are in contact with the plasmodium stain differently from those in healthy tissues,

and such cell walls are often somewhat swollen, showing a special affinity for the orange stain of the triple combination. This indicates that the cellulose is being acted on and in some manner changed, but whether the change is accomplished through the secretion of an enzyme the writer is unable to say. It is possible that such changes might be the result of the direct action of the protoplasm of the plasmodium on the cellulose.

"As the pseudopodia push down between the cells the walls of the latter seem to become somewhat gelatinous and the middle lamella is dissolved. These tapering plasmodial projections sometimes extend down into the sound tissue for a distance of five or six cell layers; often, however, they do not go deeper than two cell layers. They are irregular in shape, as might be expected from the way in which they crowd between the cells. Through these pseudopodia the plasmodium comes in contact with a large number of cells. The cell walls seem to become more and more softened and gelatinous as they thicken, and may even lose to a certain extent their original shape, becoming wavy.....

"Through the softened cellulose walls the plasmodium sends small protoplasmic strands, which may for convenience be termed the 'infecting pseudopodia'. These usually pass into the cell through openings that are quite small, but occasionally the opening is rather large..... In some manner which has not yet been determined the infecting pseudopodia become separated from the remainder of the plasmodium, and in this way the individual cells receive each a small portion of protoplasm from the plasmodium that is invading the tissue. The quantity of infecting material received by different cells is quite variable.....

"In the way which has already been described the invading plasmodium infects a small pocket of tissue just beneath the epidermis. This varies from 1 to 2 mm. in diameter and from one to six cells in thickness. Its size determines the size of the powdery-scab sorus that is to result. If this small island of infected cells could be removed, it would leave a bowl-shaped cavity covered over by the epidermis.....

"Shortly after the cells become infected, they begin to grow very rapidly. Giant cells 5 or 10 times as large as the normal ones are soon to be seen. Instead of growing equally in all directions, the infected cells elongate, most of their growth being radially outward. This results in lifting the epidermis and finally in breaking through it. The torn edges turn back on all sides and give the appearance so characteristic of powdery-scab lesions....."- Kunkel, L. O. A contribution to the life history of Spongospora subterranea. Jour. Agr. Res. 4: 265-277. June 15, 1915.

Influence of moisture and temperature on the powdery scab organism.

Ramsey (7) carried out some greenhouse experiments to determine the effect of moisture and temperature on the development of this parasite. He states that "The experiment seems to show that a cool, moist season or climate is most favorable to the development of the powdery scab organism, and that it will not develop to any marked extent, if at all, under any other conditions".

General summary.

The following quotation from a U. S. D. A. News Letter (5) sums up the situation with regard to this disease in so far as the facts are available:

"It will be recalled that when the powdery scab was first discovered in imported potatoes relatively little was known as to its nature and nothing of its behavior under American conditions. Quarantine No. 11 was a temporary and precautionary measure taken to safeguard our industry while the necessary investigations were being made. It was at first feared that powdery scab would prove more serious than common scab on account of its canker stage reported from abroad and also that, since as a rule introduced parasites have been more destructive here than at home, it might assume a very virulent form in our Southern and Western States. At that time also there was no record of its occurrence in the United States except in a limited area in Maine.

"During the past two years the following information has been secured which bears on the economic side of the problem.

"Origin.- Powdery scab has been traced to the Andean region of Peru, and is believed to be endemic in the native home of the potato. That it is a parasite of long standing is indicated by its manner of developing in the potato with the growth of the potato cells, which remain alive for some time after infection.

"Character of Injury.- Five types of injury can be distinguished:

(1) The characteristic superficial lesions, or sori on the tuber, which are only skin deep and resemble common scab as to type of injury;
(2) A shrinkage or shriveling around the spots, due to the loss of moisture, since there is less cork formation under the sori than in the case of common scab. The injury from this may be considerable depending on storage conditions;
(3) An enlargement and deepening of the affected area, due to the germination of the spore balls and inward growth of the parasite. This affects primarily potatoes carried over to spring;
(4) An active dry rot resulting from secondary infection by decay-producing fungi, usually species of Fusarium. This destroys the tubers, but is fortunately not very prevalent except under poor storage conditions;
(5) Powdery scab also attacks the roots of potatoes, tomatoes, and related Solanaceous plants and produces small galls thereon. The significance of this stage is not yet fully understood.

"Geographical Distribution.- Powdery scab occurs in eastern Canada; in northern Maine; in Clinton and Franklin Counties, N. Y.; in northeastern Minnesota, in Carlton, Lake, and St. Louis Counties; and on the Pacific coast of British Columbia, Washington, and Oregon. The most painstaking and repeated surveys have failed to show any infection south of New York City, except in one limited area in northern Florida, although it is known that many infected potatoes have been planted in the Atlantic coast section.

"Soil and Climatic Relations.- Powdery scab appears to be more or less definitely correlated with certain soil types and limited to a remarkable degree by soil and climatic conditions. It is a disease usually occurring only in poorly drained or bog soils in the most northern and humid districts. Its possibilities under such conditions may approximate the reports of serious injury published by investigators in Ireland, but for warm, light, and well-drained potato soils it is believed that the danger is small. Experimental plantings of infected potatoes have been made in nearly all the Eastern States, but no powdery scab could be found on the crop harvested. That climatic factors play a prominent part in determining the prevalence of powdery scab is shown by an experiment in which soil obtained from 10 of these States and transported to a favorable climatic condition produced diseased potatoes in 8 cases, proving that no matter what the source or condition of the soil, powdery scab will not occur unless climatic conditions are suitable.

"Control.- From the control standpoint it seems probable that the potato industry will adjust itself to powdery scab in the districts where the disease is established. Varietal resistance of a marked degree is shown by some strains. Recent tests indicate also that treatment with hot corrosive sublimate will be an effective means of disinfection and that attention to the soil reaction will have much effect.

"Determination of State Policy.- It is believed that the Central and Southern States will not need to concern themselves particularly about powdery scab, on account of soil and climatic conditions unfavorable to the disease. The measures that should be adopted to control other common potato troubles will be sufficient. The more general use of certified northern seed is especially to be desired. For the States along the northern border and the Pacific Coast States the problem is less fully settled. Further surveys should be conducted and well-controlled experiments made by the State experiment stations with powdery scab on the principal soil types to determine its reaction to the local environment."- Powdery Scab. Pathologist's Report on Study of Potato Disease-Limited by Soil and Climatic Conditions. Weekly News Letter, United States Department of Agriculture. 3: 1-5 January 26, 1916.

Review.

Describe all the different types of injury that may result from powdery scab. What parts of the host may be attacked? Give a detailed account of the life history of Spongospora subterranea. What is the present known geographical distribution of this disease? How does it rank in importance with other potato diseases? What soil and climatic conditions favor its development? What recommendations are given for its control?

References.

(1) Gussow, H. T. Powdery scab of potatoes, Spongospora subterranea (Wallr.) Johnson. Phytopathology 3: 18-19. 1913.

(2) Morse, W. J. Powdery scab of potatoes. Maine Experiment Station Bulletin 227. 1914.

(3) Melhus, I. E. Powdery scab, Spongospora subterranea of potatoes. U. S. Department of Agriculture Bulletin 82. 1914.

(4) Kunkel, L. O. A contribution to the life history of Spongospora subterranea. Jour. Agricultural Research 4: 265. June 15, 1915.

(5) Anonymous. Powdery scab; pathologists report on potato disease; limited by soil and climatic conditions. U. S. Department of Agriculture, Weekly News Letter. January 26, 1916.

(6) Osborne, T. G. B. Spongospora subterranea (Wallr.) Johnson. Annals of Botany 25: 327-341. 1911.

(7) Ramsey, G. B. Influence of moisture and temperature upon infection by Spongospora subterranea. Phytopathology 8: 29-31. January, 1918.

CHAPTER XVII.

Diseases caused by bacteria.

Some of the most destructive diseases of plants are caused by species of bacteria. Among these belong some of the so-called blights, wilts, galls, soft rots, etc. Some of the more common and serious bacterial diseases are fire-blight of apple and pear, crown-gall of various trees and small fruits, bacterial-gummosis of cherry, blight of walnut, bean blight, blackleg of potato, black-rot of cabbage, bacterial wilt of cucumber, citrus canker and a bacterial blight of oats.

FIRE BLIGHT

Caused by Bacillus amylovorus (Burr.) Trev.

This disease is probably the most serious of all pear diseases and also does great damage to apples. Practically all pomaceous fruit trees are susceptible to its attack, including pear, apple, quince and also native fruits of the same type such as service-berry and hawthorn. Occasionally it has been known to attack even stone fruits.

1. Laboratory.

a. Symptoms.- Examine specimens of diseased branches, twigs, leaves, blossoms, fruits and roots, if available, and become familiar with the symptoms of fire-blight on any part of the plant. Describe these symptoms in notes. Make drawings to illustrate. For descriptions and illustrations of these symptoms see the following: Fungous Diseases of Plants, by B. M. Duggar, pp. 122-128. Manual of Fruit Diseases, by Hesler and Whetzel, pp. 325-330. Also see the additional references listed at the end of this exercise.

b. Relation of parasite to host tissues.- Examine under the microscope, sections of both healthy and diseased pear twigs. Carefully compare the two sections. What tissues of the host are invaded by the bacterial organism? What is the effect upon the host cells? Are the bacteria between the cells, or inside the cells, or both? Draw from section to illustrate these points. Describe in notes. (See reference 9).

c. Morphology and life history of causal organism.- For description and illustrations of the organism and its life history see Fungous Diseases of Plants by B. M. Duggar, pp. 125-127. Cornell Experiment Station Bulletin 329: 326-328. Cornell Experiment Station Bulletin 372: 38-39. (See following pages).

(1) Pure cultures.- Examine pure cultures of the bacteria and describe the cultural characteristics. Make mounts and examine under the microscope. If facilities are available, stain some of the organisms.

(2) Perpetuation, dissemination and infection.- Consult the following and any other available references and find out how the disease is carried over from one season to another, how it is scattered and where and when infection takes place. Manual of Fruit Diseases, by Hesler and Whetzel, pp. 323-331. Cornell Experiment Station Bulletin 272, pp. 39-43.

d. Notes.- Write up complete notes embodying all information obtained from any source upon this disease.

2. Recitation.- Before the class meets for recitation, read as many of the following references as possible and be prepared to discuss in detail the symptoms, life history and control of this disease.

Text discussion.

The following discussion of names, occurrence, distribution, economic importance, and symptoms is quoted from Stewart (4):

"Names.- The disease has been a theme for incessant discussion by horticulturists since the earliest days of fruit culture in this country. Various common names have been applied to the disease, depending to some extent on the host that is attacked and on the effect produced. Among the names most frequently used are fire blight, pear blight, blight, blossom blight, twig blight, fruit blight, sun scald, canker, and blight canker. The term "fire blight" is preferable, since it applies equally well to the characteristic symptoms of the malady on any of the different hosts.

"Occurrence.- Occurring as a disease of the indigenous wild crab-apple and hawthorn of eastern North America, the blight spread readily to orchards of pome fruits when the cultivated varieties were introduced into this country. It was first observed about 1780 in the Hudson River Highlands of New York State by William Denning (1794), an orchardist of that section. Later, with the increase of the orchard industry, the disease became more prevalent and gradually worked its way westward, as the plantings followed the settlement of the country beyond the Allegheny Mountains.

"Geographical Distribution.- The disease occurs throughout the United States and Canada in practically every section where pomaceous fruits are grown. No authentic records have ever been made of its occurrence in Europe or on any of the other continents. Its restriction to this country may be attributed, no doubt, to the fact that the quantity of stock of pomaceous fruits exported from America is comparatively small, thus eliminating to a considerable extent the possibility of the disease being transported across the ocean.

"Fire blight is common in all the nursery districts of New York State and has frequently been destructive in the plantings of the Southern States, especially Alabama, Florida, and Georgia. It is a well-known malady in the nurseries of the Central and Middle West and has been reported by Orton ('02) as being especially abundant in Texas and Louisiana. The disease is somewhat generally distributed over the pear-growing section of the Pacific Coast, particularly in California and Oregon.

"Economic Importance.- Without question, fire blight is the most important disease affecting the pomaceous fruits. Being of an epidemic nature, it may suddenly appear in a locality with increased prevalence and cause complete destruction or severe injury to the fruit-tree industry of that section. Pear orchards, especially, are severely attacked, and often an orchard is entirely destroyed in one season. Although the blight may have subsided as an epidemic, it is usually found to a limited extent in any locality and under conditions favorable for its development and propagation it again becomes a destructive disease. Usually, in the nursery it means total loss to the trees affected, and with its rapid spread through the blocks where the trees are thickly planted, often within a comparatively short time, thousands of young trees are ruined. In some cases entire blocks of apples, pears, and quinces have been destroyed. Such a condition prevailed in one of the nursery districts of New York State in 1908, and practically every nurseryman in that section suffered heavy losses from the disease.

"Symptoms.- The limbs, blossoms, twigs, and fruit may be attacked. In the nursery the disease is most commonly found affecting the twigs, one exception being the two-and three-years old quince stock. These trees often blossom profusely in the spring, and under this condition blossom blight frequently occurs.

"Blossom blight. Sometime in the early part of the season, about two or three weeks after the blossoming period, the blight first attracts special attention and by a close examination the blighted blossoms may be detected even somewhat earlier. The first evidence of the trouble is the brown and subsequent blackened appearance of the young leaf tufts and the blossoms, from which the disease rapidly extends into the fruit spurs. It prevents the development of the fruit and may even involve the larger twigs and branches.

"Twig blight.- The wilted and brown or dead appearance of the stem and foliage is the characteristic symptom of twig blight. The progress of the disease down the stem, in the early stages, may be in advance of any marked discoloration of the foliage, and to a considerable degree the blighted twigs resemble green brush that has been only partially burned. There is generally a viscid, milk-white substance exuding in small drops on the surface of the twig or the petioles, which later becomes oxidized into an amber-yellow or slightly brownish, then finally into a dark brown or almost black, gum.

"The extent of the disease in advance of any discoloration of the foliage may be determined by the sappy and juicy appearance of the tissue. A faint amber-yellow or reddish discoloration of the tip is often a means of detecting recently affected apple shoots. In pear twigs an intensive blackening of the tissue is usually characteristic, even in early stages of the infection. However, in all host plants the blight ultimately produces the same effect--the leaves shrivel, turn brown or black, and resemble foliage that has been killed by frost. One of the most striking symptoms of fire blight to be recognized is in the twig or limb with dead, brown or black leaves clinging to it, contrasting sharply with the dark green foliage in summer and the naked branches of the trees in winter. In no other disease of the pome fruits do the leaves cling so tenaciously to the dead twigs.

"Body blight.- The blight often works down the twigs or branches into the trunk of the tree and within a short time may extend into the roots, causing the destruction of the whole tree. While the disease is active in the trunk of the larger branches, the affected tissue is darker in color, often with a brownish or reddish tinge. The abundance of sap gives the bark a water-soaked, or occasionally a slightly raised and blistered, appearance. (The blisters have also been observed even in the smaller pear and quince twigs.) The characteristic exudation is common and, when the disease is especially active, it may be so abundant that it flows slowly down the side of the tree.

"With unfavorable conditions--as, for example, at the termination of the growing season, when the plant tissues harden and there is a diminution in the sap supply--the active progress of the blight becomes checked by natural causes. The diseased bark shrinks and subsides, resulting in a sharp line of demarcation between the healthy and the diseased tissues.

"When only a definite area is affected, the diseased part surrounded by healthy bark is known as a canker. The inner tissue of the diseased bark appears brown or dead, making a distinct contrast with the pale green or white, healthy tissue. With favorable conditions the following spring it again becomes active and progresses farther into the adjoining healthy bark. Hold-over cankers, in which the blight remains in a latent form throughout the dormant season, are rather common."- Stewart, V.B. The Fire Blight Disease in Nursery Stock. Cornell University, Agricultural Experiment Station Bulletin 329. April, 1913. Pages 318-323.

Collar-blight and related forms of fire-blight.

Orton and Adams (6) have given the following description of this disease as it sometimes occurs at the base of the tree:

"The forms of disease of the apple, pear, and quince caused by the fire-blight organism are the most serious troubles with which the Pennsylvania apple grower has to contend. That form of blight which attacks the trunk of the tree near the ground, and which may be known as collar-blight, is especially troublesome.....

"That the disease is not a local one is shown by references to it in the published reports and bulletins of several State experiment stations and it would seem now to be rather widespread in New York, New Jersey, Pennsylvania, Maryland, Virginia, West Virginia, Ohio, Illinois, and Oregon. It probably occurs to some extent in nearly all of the apple-growing sections of the United States. What is apparently collar-blight is referred to by Selby in Ohio as destructive to Grimes Golden, Tompkins King, and other varieties of apple. Whetzel refers to collar-blight in the following words: 'The large cankers at the bases of young trees, frequently referred to by growers as 'collar-rot', are in many cases very probably due to the same causes as that of the cankers on the upper parts of the trees.'....

"R. E. Smith refers to collar-blight in California as occurring in orchards where fire-blight is present and where, as he says, 'it appears that certain roots have been badly injured and that the portions affected above ground correspond with such roots'.

"Jackson (5) refers to the prevalence of collar-blight in Oregon stating that, 'it is this form of the disease that causes many trees to be killed outright'.

"Giddings reports that collar-blight is one of the most serious diseases of the apple in West Virginia.....

"The early symptoms of collar-blight are usually evident during the growing season by a more or less unhealthy appearance of the foliage. The leaves appear yellowish and smaller on one or more of the large limbs as contrasted with normal foliage. When infected areas on the trunk progress slowly and extend through two or more seasons, the foliage, especially on that side of the tree affected, is rather sparsely developed. Since this effect on the foliage is rather gradual it does not result in the wilting or drying of the leaves in contrast to fire-blight infection of twigs. The ultimate effect on the foliage in all cases is the premature falling of the leaves.

"The general yellowing of the foliage facilitates the recognition of infected trees during the growing season. On examination of the collar of such trees there will be found a sunken area of variable size depending upon the time which has elapsed since infection took place. Newly infected areas are not sharply marked off as in the more advanced cases. The disease progresses rapidly until midsummer when its development is arrested. During the next season activity is renewed, the attack often resulting in the complete girdling and subsequent death of the tree. The girdling at the collar may be said to be the usual method of development but often, under certain favorable conditions not yet ascertained, the blight may extend very rapidly up one side of the trunk into the branches, and thus cause sudden death. In some cases it has been observed to extend downwards, thus affecting the lateral roots for some distance.

"Usually it is not until the second season of infection that the characteristics of the disease above the ground are clearly defined. A distinct line of demarcation separates the normal tissue from the lesions, which appear to be water-soaked and browned, eventually separating from the sap-wood. The diseased tissue emits a sour odor and often small milky drops exude which consist of masses of bacteria. Pure cultures of Bacillus amylovorus have often been isolated from these exudations. The sap-wood becomes browned and later blackened.

"During the second season the foliage is usually scanty, and it is common for such trees to blossom heavily and to set fruit which develops under size and inferior in quality with a tendency to fall early. Attempts at healing which have prevented complete girdling have been observed often. In some cases of apparent normal healing, further extension of the infected area has been caused by borers working from the old lesions through the callus into the uninfected region.....

"From the nature of the causal organism, and the long-tried methods for its control by surgical treatment, it has seemed that the application of such methods might be of practical use in treating trees blighted at the collar. On this assumption the junior author carried out a series of experiments in 1912, to ascertain whether collar-blight might be successfully checked by cutting out the diseased areas. Trees in all stages of the blight were chosen and from the results

of these experiments it would seem possible to cut out collar-blight with good chances of saving the trees, provided they are not more than half girdled. If more than half girdled, it hardly seems advisable to attempt to save the tree unless bridge-grafting can be resorted to advantageously."- C. R. Orton and J. F. Adams. Collar-Blight and Related Forms of Fire-Blight. Pennsylvania Experiment Station Bulletin 136. August, 1915. pages 16-17.

Life cycle of the organism.

Stewart (16) gives the following description of the life history of the fire-blight germ:

"The bacteria remain alive throughout the winter in the body, the limbs, or even the twigs of the tree. With the ascent of the sap and the increased temperature of spring, the organisms in the so-called "hold-over" cankers become active again, multiply, and spread into the adjoining healthy bark. Often, when their increase is augmented by warm rainy weather the gummy exudation, laden with bacteria, oozes out of the lenticels and cracks of the infected bark. This exudation, containing millions of the minute bacteria, usually occurs first about the time when the blossoms are opening. Various insects such as wasps, honeybees, and flies, are attracted to the hold-over cankers by the sweet, gummy exudate, become smeared with it, and carry the infection material to the blossoms. Some of the bacteria brought by the infected insect are left in the nectar of the flower. The sweet solution greatly favors the multiplication of bacteria, and the next bee or other insect carries the organism to all succeeding blossoms that are visited. The bacilli penetrate the tender tissues of the flower and within nine or ten days the characteristic symptoms of blossom blight are apparent.

"After the blossoming period, or sometimes even before it is entirely over, the blight may appear in the young, succulent shoots or twigs. Ordinarily in the nursery, where blossoms are not common except in the two-and three-years-old quince stock, the first occurrence of the blight in the spring is in the twigs. Instances have been observed, however, when the disease appeared first in the quince blossoms and spread later to the tender shoots. Not only do the new twig infections continue to occur throughout the summer, but the disease continues to spread in an affected tree and, unless checked, may involve the entire tree.

"As a general rule the blight is first found in the two-and three-years-old trees; later, these trees are the source of infection for the spread of the disease to the first-year buds. The insects that disseminate the blight bacteria, such as the tarnished plant bugs, are most abundant on the apple stock throughout the months of July and August, and usually the blight in the first-year buds is most severe at this time.

"Disseminating agents.- Such agents as bees and wasps have long been conceded important in the spread of blossom blight, but in recent years certain other insects have been associated with the spread of the disease, particularly with twig blight. While visiting blighted tissues the insect becomes smeared with the gummy exudate and carries the bacteria to the tender twigs. In sucking the sap from the twigs the insect punctures the tissue and thus furnishes a means of entrance for blight germs.

"The common green aphids (plant lice) frequently spread the disease in apples and quinces. However, of all the sucking insects that may disseminate the blight bacteria in the nursery, perhaps the most important is the tarnished plant bug (Lygus pratensis). This insect has been recognized for many years as the cause of the severe injury to peach stock known as peach sting. The injury resulting from the insect's 'stinging' the terminal shoot of the first-year peach buds causes the tree to make a stunted, bushy growth and to fall short of the proper height. During the past two seasons, not only has the peach stock been injured in this way, but considerable damage has been apparent in several blocks of first-year apple buds. A large number of apple shoots that had been stung by insects a few days previously developed infections of fire blight. When abundant in blocks of two-years-old stock (especially in apples where there may be considerable blight) the insects visit the sweet, gummy exudation on infected trees, and thus not only spread the disease to the large trees but carry the bacteria to adjoining blocks of one-year-old apple stock where infections occur through the punctures made by the insects.

"Dissemination by Pruning Tools.- Besides the general distribution of the blight germ by insects, man himself is often an active agent in spreading the parasite. His pruning tools are certainly a frequent means of transmitting the organisms. M. B. Waite, of the United States Department of Agriculture, writes: 'In Maryland the writer once saw a nursery block of 10,000 trees of Bartlett and other pears completely destroyed by blight. This block, as determined by the specimen, carried actual samples of hold-over blight in the stocks. When the stocks were cut off above the dormant buds in the spring, the pruning tools became infected and the disease was transmitted to nearly every tree reached by the pruners. Instead of the buds pushing up, the cut surfaces began to gum and blight.

"Blight is frequently introduced into seedling blocks by the use of diseased scions that have been cut from two-and three-years-old stock in which considerable blight has been present. The seedlings, which are budded with diseased buds, not only blight, but bacteria are carried on the budder's knives to other seedlings. Later, at rebudding time, the budders, being unfamiliar with the disease, frequently attempt to rebud the blighted stocks; their knives become infected and the bacteria are transmitted to other seedlings. The number of diseased seedlings is also frequently increased when the strings used for tying the buds are cut. Often in cutting the string a slight incision is made in the bark and the trees are inoculated in this manner, blight germs having been carried on the knives from diseased stocks.

"Blight may also be introduced into the seedling block by means of insects, which carry the bacteria to the tender shoots. Occasionally, entire trees become diseased in this manner. However, twig infections are seldom if ever so abundant in the seedlings as to be the source of a serious outbreak of the disease. On the other hand, an attempt to bud such infected seedlings would tend to favor the more rapid spread of the disease, the blight bacteria being carried to healthy trees on the budding knives.

"The shipment of scions from one nursery to another may also be a means of transmitting the blight for long distances, and bacteria have even been known to live over in scions used for grafting purposes. In the winter, when the grafts were made, the grafting knives became infected and transmitted the blight bacilli

to grafts made from healthy scions."- Stewart, Vern B. The Fire Blight Disease and Its Control in Nursery Stock. Cornell University, Agricultural Experiment Station Circular 20. May, 1913. pages 89-91.

Some very valuable side-lights on the dissemination of fire-blight bacteria are found in the following summary from Ohio Bulletin 357, by Gossard and Walton (14):

"1. Our investigations have proved that the organism of fire blight may live in honey for 72 hours or more, but this approaches the optimum limit.

"2. We found that fire blight developed in tender twigs that were inoculated with honey drawn from three different hives. From two of the twigs thus blighting, the organism was recovered and by reinoculation into other twigs was proved to be fire blight.

"3. By using apple pollen removed from the baskets of bees caught as they were entering the hive to inoculate into tender twigs, we obtained several cases of death, apparently from fire blight, but the organism was not recovered nor its identity proved.

"4. By inserting the mouthparts of bees, caught during blossoming, when entering their hives, into incisions into tender twigs, we obtained some twigs which seemed to have died without doubt from fire blight, but the organism was not recovered nor its identity proved.....

"6. The fire-blight organism was proved to be able to live in aphid honey-dew for 7 full days until the dew was completely dried and then for 3 more days or perhaps longer when moisture was added.

"7. The organism was also proved to be able to live in peach, plum, and cherry nectar for 5 full days or probably more and capable of producing blight in apple twigs when inoculated into them.

"8. Rain was proved to be a most important carrier of infection over a tree after centers were established, especially if near the top. We estimated that from 50 to 90 percent of all blossom infection is accomplished by infected rain water.

"9. We proved that blossoms which have been pollinated for 72 hours are not likely to be inoculated with fire blight and that susceptibility to inoculation does not exist in blossoms that have been pollinated for 144 hours.

"10. We infer from number 9, that numerous honey bees in an orchard to fertilize the blossoms as soon as they open will insure a crop of fruit by putting the early blossoms past susceptibility to blight before the disease is generally diffused over the orchard.

"11. The initial infection of a young pear orchard was observed in one case to have originated through blossom blight.....

"12. Efforts to prove that ants were responsible in a large way for early spring infection met with negative results.

"13. Attempts to isolate the blight organism in early spring from the bodies of aphids, aphid wax and syrphid larvae taken from living blight cankers met with negative results.

"14. We submit that there is ample ground for crediting blossom fire blight with traveling northward with the zone of inflorescence to a greater extent than is now accepted.

"15. Branches covered with cheesecloth bags to exclude insect pollinators set fruit for only 1.36 percent of the blossoms while 18.58 percent of the blossoms set fruit on uncovered branches....."

Miscellaneous.

Hotson (10, 17) has made some interesting observations on fire-blight in the state of Washington. Three items from his articles are quoted below:

"Fire Blight on Pear Leaves.- An interesting case of leaf infection has been repeatedly observed on different varieties of pears, especially the Bartlett. This was particularly evident after a rain of which there were two unusually heavy ones during the early summer. This condition on leaves was first observed towards the end of June but it undoubtedly existed earlier in the spring than that. The infected leaves turn brown in sections, the brown areas always including a portion of the margin of the leaf. In some cases only a small portion of the leaf, extending in from the margin a quarter or half an inch, is affected while in others the affected area extends to the midrib, and in still others spreads nearly or completely over the whole leaf.....In only a small number of instances were the leaves completely discolored or any evidence shown that the infection ran back through the petiole into the branch.

"At first this condition was considered a sun-scald, but occasionally leaves were brought to the laboratory with exudate on them, particularly, as has been said, after a rain..... A microscopic examination of this exudate showed that there was a large number of bacteria present, which suggested fire blight. To determine definitely if these discolored areas were the result of Bacillus amylovorus, tube cultures were made from the exudate and also from infected pieces of leaves..... As a result of these experiments, over ninety per cent of the cultures taken from the exudate readily produced the characteristic fire blight colonies on sterilized pears.....

"As far as the writer is aware this represents an entirely new mode of attack by the fire blight organism. It would appear that the bacteria gain entrance either at the margin or through punctures in the leaves near it. The fact that invariably a portion of the margin is included in the discolored area would tend to point to it as the place of entrance. This theory is emphasized by the fact that not infrequently the margin appeared to have been injured, often having a small crack or split through which the organism may have entered. There were, however, not a few instances in which no evidence of an abrasion or puncture could be detected on the leaf when carefully examined with a hand lens."- Hotson, J. W. Observations on fire blight in the Yakima Valley, Washington. Phytopathology 6: 288-289. June, 1916.

"Fire Blight in the Sapwood.- Another interesting condition has been observed in connection with fire blight in the Yakima Valley, on the Comice pears and less frequently on Winter Nelis and Bartlett pears. On several occasions, and from widely different sources, the writer's attention has been called to a condition occurring on limbs and twigs infected with fire blight where the sapwood and sometimes the pith showed a decided reddish color similar to the red streak that is characteristic of fire blight.....

"An effort was made to determine whether this peculiar condition was caused by the blight organism or merely associated with it. Many examinations of twigs and branches were made, the red streak always being found on limbs that were or had been blighted and never on perfectly sound, normal branches. From this fact it was concluded that this condition was due directly or indirectly to fire blight. Gross cultures and also tube cultures were made of the red sapwood with varying results.....

"It would appear from these observations and experiments that under certain conditions of terminal or fruit-spur infections, the bacteria may invade not only the bark but the young sapwood and even the pith."- Hotson, J. W. Observations on Fire Blight in the Yakima Valley, Washington. Phytopathology 6: 289-290. June, 1916.

"The Longevity of Bacillus Amylovorus Under Field Conditions.- The object of these experiments was to obtain, if possible, some definite information regarding what actually occurs in the orchard, how long fire-blight organisms live in the dried-up exudate; how long they live in infected branches that are left under the trees, in deep grass, or in clear cultivation; and also the effect of sunlight on the organisms in cankers that have a thin outer layer of the bark removed.....

"These experiments clearly show that the fire-blight organism (Bacillus amylovorus) under field conditions, at least under those found in the Yakima Valley, lives longer than has been generally supposed from work with artificial cultures. The reason for this is readily seen in the protection these organisms obtain from the bark of the tree and the hardened gelatinous envelope caused by the drying of the exudate. The process of killing is often although not always, simply that of desiccation. The larger the limbs that are cut off and the larger the beads of exudate the longer does the organism retain its vitality. In a similar way the amount of moisture surrounding the blighted branch after it is cut off determines to a large extent the length of time the organisms will live. This is especially true where the temperature is reasonably low, from 15°C. to 18°C. If blighted branches are cut off and left in a covercrop of clover or alfalfa the protected organisms may retain their vitality a month and at the time of irrigation or rain the exudate may soften and begin to ooze, thus becoming a source of further infection. Unless the branches are thus moistened they may not exude and therefore may not be a source of infection, but it is wisest to burn the branches as soon as possible after they are cut off."- Hotson, J. W. The longevity of Bacillus amylovorus under field conditions. Phytopathology 6: 408. October, 1916.

Control.

There are three means of combating this disease. (a) Cutting out the diseased parts, disinfecting both the wound and the pruning tool after every cut. (b) Controlling the disseminating insects. (c) Selecting and developing resistant or immune varieties.

Cutting out

The common disinfectant in use for this purpose has been bichloride of mercury (corrosive sublimate). Recently Reimer (12) has discovered a much more effective disinfectant for this purpose. An account of his experiments follows:

"A New Disinfectant For Pear Blight.- It is generally known by pear growers that the only way to eliminate pear blight (Bacillus amylovorus) from an infected tree is to cut out and destroy all the infected parts of the tree. It is also well known that in cutting out and through these infected portions often many blight bacteria are smeared over the tools used in the work. Furthermore when the final cuts are made in the clean healthy bark some of the bacteria on the tools again are smeared over the healthy surface. In many cases new infections inadvertently are made in this manner, and the disease continues to spread from the margins of the wound. For this reason a disinfectant should be applied to destroy any bacteria that in this way may have been left on the surface of the wound. The only disinfectant that has been recommended generally and used widely for this purpose is corrosive sublimate (bichloride of mercury). This is a very powerful disinfectant and has been extensively used in the past by medical men.

"It often has been noted that where the blight cankers have been removed and corrosive sublimate applied, the disease would continue to develop, especially during favorable weather conditions. This has been attributed generally to a lack of thorough work, leaving some blighted tissue in the margins of the wound. It is certain that this has been responsible for such continued development of pear blight in very many cases.

"However, often pear blight would continue to develop from such wounds where the most thorough work possible had been done. For this reason it was suspected that possibly corrosive sublimate was not destroying all of the bacteria left on such wounds. Hence experiments were started in June, 1918, to test the efficacy of corrosive sublimate and other chemicals as disinfectants for pear blight germs on the wounds of pear trees.

"For the first experiment a large number of uniform and very vigorous three-year-old Bartlett pear trees were selected. These were entirely free from pear blight at the time the experiment was started. On the trunk of each tree two large wounds were made by removing the bark and exposing the sapwood over the entire wound. Blight bacteria were then smeared over the surface of each wound. especially over the margins, by which process most of the bacteria were deposited on the cut surface of the margin of each wound. In order to preserve uniformity all of the bacteria utilized were taken from one culture.

"Immediately after placing the bacteria the disinfectants were applied, thoroughly covering the entire wound by means of a new, clean, paint brush. For each disinfectant a separate brush was used.

"The following disinfectants were used, treating with two exceptions eight trees or sixteen wounds with each disinfectant: (1) Bordeaux paste; (2) corrosive sublimate, 1 to 500; (3) cresol 5%; (4) cyanide of mercury, 1 to 500; (5) lime sulphur, 10% (6) "black-leaf 40", 5% (7) chlorozene, 1 to 500. Eight trees in which the bacteria were applied to the wounds but no disinfectant was used, were utilized as checks.

"The results of this experiment were rather startling. Every wound treated with corrosive sublimate, Bordeaux paste, cresol, lime sulphur, black-leaf 40, and chlorozene developed a vigorous case of pear blight. The corrosive sublimate was used twice as strong as is recommended generally for this purpose, and the brand used is manufactured by one of the largest and best known chemical firms in this country. The Bordeaux paste was made by dissolving one pound of bluestone in one gallon of water, and two pounds of lime in one half gallon of water, and then mixing the two. The cresol used was the liq. cresolis compositus. The lime sulphur was one of the best known commercial brands and tested 32° Baumé. Chlorozene is the new disinfectant now used extensively in treating human wounds.

"It is well known that corrosive sublimate is a very powerful disinfectant under certain conditions. In pure water cultures it will kill readily most bacteria in a comparatively short time when used at a strength of 1 to 1000. It has been known for a long time that its value is impaired by organic matter, and especially by albumins. In fact in the presence of large quantities of organic matter it forms inert combinations which reduce, if not totally destroy, its value as a disinfectant.

"This probably explains our results with corrosive sublimate as a disinfectant on the fresh wounds of pear trees. These wounds are largely polluted by organic matter, and under these conditions corrosive sublimate is not thoroughly effective.

"Results With Cyanide of Mercury.- The treatment with cyanide of mercury, in the foregoing experiment, proved very effective. Not a single treated wound developed blight. In this experiment pure cyanide of mercury was used at a strength of 1 to 500 (1 gram of pure cyanide of mercury to 500 grams of water). It is remarkable that in all the other treatments blight developed whereas in the case of the cyanide of mercury not the slightest indication of the disease could be found. The margins of the wounds showed some injury from the treatment in every case where the cyanide of mercury was used. This injury was confined to a narrow strip of bark around the wound, and was of no special importance as the cambium soon started to push out from underneath the injured bark and continued to grow out over the wound just as it does in normal wounds where no injury has been produced."- Reimer, F. C. A New Disinfectant for Pear Blight. Monthly Bulletin of the State Commission of Horticulture of California. 7: 562. October, 1918.

Resistant or immune varieties.

Reimer (13) has made several trips to China and has brought home several species of pears, some of which have proved highly resistant, or practically immune in a few cases. He is using these for grafting and budding stock and meanwhile is carrying on hybridization work in hopes of getting a variety that will be immune and at the same time be of high commercial value.

Review.

Name all the various parts of the plant which may be attacked by fire-blight and describe the effects of the disease on each part. Describe the manner of perpetuation, dissemination and infection. What have climatic, seasonal and weather conditions to do with the spread and seriousness of this disease? How does the disease spread within the host? In view of what has been learned about the nature of this disease and the life history of the causal organism, discuss all methods and devices which might be of practical use in the control of fire-blight in the orchard. Look up the question of susceptible and resistant species and varieties.

References.

(1) Duggar, B. M. Fungous Diseases of Plants, pp. 121-129.

(2) Hesler & Whetzel. Manual of Fruit Diseases, pp. 232-331.

(3) Whetzel, H. H. and V. B. Stewart. Fire-blight of pears, apples, quinces, etc. Cornell Experiment Station Bulletin 272. 1909.

(4) Stewart, V. B. The fire-blight disease in nursery stock. Cornell Experiment Station Bulletin 329. 1913.

(5) Jackson, H. S. Fire-blight of pear and apple. Oregon Experiment Station. Cir. Bul. 7, 1913.

(6) Orton, C. R. and J. F. Adams. Collar-blight and related forms of fire-blight. Pennsylvania Experiment Station Bulletin 136. 1915.

(7) Stewart, V. B. and M. D. Leonard. The role of sucking insects in the dissemination of fire-blight bacteria. Phytopathology 5: 117. April, 1915.

(8) Heald, F. D. Preliminary note on leaf invasion by Bacillus amylovorus. Washington Experiment Station Bulletin 125. 1915.

(9) Bachman, Freda. The migration of Bacillus amylovorus in the host tissue. Phytopathology 3: 3. February 1913.

(10) Hotson, J. W. The longevity of Bacillus amylovorus under field conditions. Phytopathology 6: 400. October, 1916.

(11) Reimer, F. C. A new disinfectant for pear blight. Monthly Bulletin Calif. Commission of Horticulture, 7: 562. October, 1918.

(12) Reimer, F. C. A new and effective disinfectant for pear blight. Better Fruit, volume 13, No. 10, pp. 24-27. April, 1919.

(13) Reimer, F. C. Some promising new pear stocks. Calif. State Commission of Horticulture, Monthly Bulletin 5: 166. May, 1916.

(14) Gossard, H. A. and R. C. Walton. Dissemination of fire-blight. Ohio Exp. Sta. Bul. 357. 1922.

(15) Smith, E. F. Bacterial diseases of plants. pp. 359-388.

(16) Stewart, V. B. The fire-blight disease and its control in nursery stock. Cornell Univ. Exp. Sta. Cir. 20. 1913.

(17) Hotson, J. W. Observations on fire-blight in the Yakima Valley, Washington. Phytopathology 6: 288. June, 1916.

CROWN GALL

Caused by Bacterium tumefaciens Smith & Townsend.

Crown gall is a bacterial disease of plants which is characterized by gall-like or wart-like overgrowths or tumors occurring on almost any part of the plant but quite commonly found just beneath or at the surface of the ground. It attacks all kinds of fruit trees and smaller fruits, as well as a great variety of other plants, wild and cultivated. The causal organism, instead of killing the invaded tissues, as is the case in fire-blight, for example, stimulates the cells to rapid division, thus producing an enormous number of small cells and resulting in an overgrowth of the diseased part.

1. Laboratory study.

a. Symptoms.- Make a study of symptoms from specimens available. Note the different types of the gall; underground form, aerial form, and hairy-root form. Draw and describe. For illustrations and descriptions of these various types of crown gall see the following: Duggar, B. M. Fungous Diseases of Plants, pp. 114-118. Hesler and Whetzel, Manual of Fruit Diseases, pp. 108-112. Smith, E. F. Bacterial Diseases of Plants, pp. 413-472.

b. Effect upon the tissues of the host.- From the specimens and from the literature cited determine the effect of the parasite upon the cells and tissues of the host. Of what kinds of tissues does the tumor consist? See the following

references: E. F. Smith. Bacterial diseases of Plants, pp. 424-431. U. S. Department of Agriculture, Bureau of Plant Industry, Bulletin 213. 1911. U. S. Department of Agriculture, Bureau of Plant Industry Bulletin 255. 1912.

c. Life history of causal organism.- Look up the facts concerning the life history in your text and the other references cited. Learn how perpetuation, dissemination and infection are effected, also look up the manner of spreading within the host by means of tumor strands.

d. Notes.- Read as many of the references listed below as time will permit and then with the information thus gained in addition to that learned in the laboratory, write a complete account of the crown gall disease.

2. Recitation.- Use the references and quotations listed below in preparing for the next recitation.

Text discussion.

During the first two decades of the twentieth century Erwin F. Smith and his co-workers did a great work in clearing up some of the problems connected with crown-gall. More recently other workers have taken up the work and added to our knowledge of this disease, clearing up some points that Smith perhaps failed to diagnose correctly.

The following from Bureau of Plant Industry Bulletin 213, 1911, gives a summary of some of the earlier work done on this disease by Smith and his helpers (5).

(1) "Crown-gall is a disease common in nurseries on the roots and shoots of various plants, and likely to continue on the plants when they are removed to orchards, vineyards, gardens, and hothouses. It also occurs on various field crops. This name is used for the disease whatever the situation of the galls on the plant.

(2) "When we began our studies the cause of crown-gall was unknown, and by them it has been determined.

(3) "Bacteria were seen in crown-galls of the daisy in 1904, and the studies then undertaken have been pursued continuously to date, and are here first offered in complete form.

(4) "The first successful isolations and infections were obtained in 1906, and the biology of the bacterial organism derived from the daisy has been determined more carefully than that from galls on other hosts.

(5) "Hundreds of pure-culture inoculations on daisy have removed the subject from the domain of speculation and shown that the galls on Paris daisy are due to a white schizomycete named Bacterium tumefaciens (April, 1907).

(6) "This organism is a short rod multiplying by fission and motile by means of polar flagella. It can be grown in many sorts of culture media, but does not live very long upon agar. It forms small, round, white colonies in agar or gelatin poured plates. Under unfavorable conditions of growth it readily develops involution forms.

(7) "This schizomycete differs from many bacterial organisms in not producing open cavities in the plant. It appears to occupy the living cells in small quantities causing rapid proliferation.

(8) "We have not been able to stain it in the tissues, at least not satisfactorily.

(9) "It is readily plated from young sound galls, i.e., those not fissured or decayed, often in practically pure culture, but it comes up slowly on + 15 nutrient agar, and generally not very abundantly. It grows however, promptly on agar when transferred from cultures.

(10) "It produces galls most readily in soft, rapidly growing tissues. Ordinarily, resting tissues can not be made to produce galls. Turnips seem to be an exception.

(11) "Cross-inoculations to plants of other families have shown the daisy organism to be capable of inducing tumors on many species in widely separate parts of the natural system (Compositae to Salicaceae), these galls varying somewhat in appearance.

(12) "Some species of plants were not infected (onion, fig, olive) and possibly are not infectable, but further experiments should be made.

(13) "For purposes of comparison natural galls have also been studied on the following plants: Peach, apple, rose, quince, honeysuckle, Arbutus unedo, cotton, poplar, chestnut, alfalfa, grape, hop, beet, salsify, turnip, parsnip, lettuce, and willow.

(14) "From all of the preceding, by means of Petri-dish poured plates on agar, schizomycetes have been isolated closely resembling (as grown on agar) the Bacterium tumefaciens obtained from the Paris daisy.

(15) "With eight of these organisms tumors have been produced on sound specimens of the species from which obtained. With these eight and two others (not tested on the host) tumors have been produced on daisy and various other plants, thus tending to show a wide range of natural cross-inoculability.....

(16) "The beginnings of the galls are visible in some cases as early as the fourth day after inoculation by needle prick, and they often reach a large size in one to two months, but frequently on woody plants they continue to grow for several years. On the contrary, sometimes they have been very slow to develop.....

(17) "The galls are often rapidly invaded by saprophytic bacteria especially the softer galls. On agar poured plates many of these bacteria are readily distinguished from the parasite by differences in form and color, but others are distinguished therefrom with great difficulty, cultures on other media or inoculations being requisite.

(18) "The galls also invite various parasites - nematodes, fungous root rot, fire blight of apple and pear, etc., and some of these are able to cause great damage.

(19) "We have not been able to distinguish etiologically between hard galls and soft galls. Even the hardest crown-galls are due to bacteria which closely resemble those found in the softest.

(20) "Overfed plants are more subject to the disease than those making a moderate growth.

(21) "The size of the tumor, other things being equal, depends on how rapidly the plants are growing, i.e., their state of nutrition. Actively growing plants usually developed large tumors when inoculated, and slow-growing galls may finally become large. This long-continued growth would not be possible if there were not a very nearly even balance between the stimulus of the parasite and the response of the host.

(22) "The apple hairy-root, hitherto a disease of unknown origin and supposed to be noninfectious, has been shown to be due to bacteria which culturally and morphologically differ, if at all, only slightly from the crown-gall organisms.

(23) "This causal organism is located not in the hairy roots themselves but in the flattened tumor from which such roots arise.

(24) "Typical hairy-root has been produced on sound apple seedlings by pure-culture inoculations, and in the same way on sugar beet both galls and hairy-roots have been obtained.

(25) "These abnormal growths which we have designated indifferently as tumors or galls are believed to be like malignant animal tumors in various particulars: Permanent and very rapid new growth containing all the tissues of the part attacked; enormous round-celled or spindle-celled hyperplasia; great reduction of amount of conductive tissues; early necrosis, especially of the more fleshy tumors, with renewed growth at the margins; frequent recurrence after extirpation; extension of the disease to other parts by metastases, etc.

(26) "The disease is one which progresses slowly, stunting the plant first and finally destroying it, unless removed by extirpation or by the development of increased resistance on the part of the plant.....

(27) "The organism is moderately susceptible to germicides but can not be reached in the galls....."

Smith, Erwin F., Nellie A. Brown, and C. O. Townsend. Crown Gall of Plants: Its Cause and Remedy. Bureau of Plant Industry Bulletin 213. February 28, 1911.

Tumor strands.

In 1912 Smith published another bulletin (6) on crown-gall in which he reiterated with more emphasis the statement made in his previous article quoted above to the effect that there is a strong resemblance between crown-gall and cancer in the animal kingdom. He makes a special point of the "tumor strand" theory. The following quotation gives his conception of this phenomenon:

"Soon after the appearance of a primary tumor, particularly if the plant is well nourished and growing rapidly, tumor strands push out of it into the normal tissues, generally, it would seem, along lines of least resistance. These, of course, are invisible externally, but may be found by dissecting the basal parts of the tumor, and if of any size they are readily detected without the use of the microscope, i.e., by their peculiar structure and color (daisy). Subsequently when they are extending in thin leaves they may be recognized sometimes by a slight tumefaction on the midrib or leaf vein, which ceases beyond the advancing tip of the strand; and especially by the development on the strand of secondary tumors hidden at first by the overlying normal tissues of the leaf but soon giving to the latter a puffed-up appearance and subsequently coming to the surface of the plant by crushing and rupture of the overlying parts. Secondary tumors are a very common phenomenon in the Paris daisy and always, so far as observed, they are outgrowths from the deep-lying tumor strand, which is itself an outgrowth of the primary tumor.

"This tumor strand in the Paris daisy makes its way exclusively, so far as observed, in the protoxylem region of the plant, i.e., in the region of the primitive spiral vessels, where it is often under great pressure, especially in the stem. So far as known, the tissues under pressure are not absorbed, but they are often flattened, crushed, split open, and exfoliated by the growing tumor. In a tobacco stem a tumor strand was observed in the bark parenchyma. In a number of instances we have found short tracheids in process of development directly from cells of the tumor strands, their lignification being yet incomplete and their nuclei still present."

Location of the bacteria.

Smith claims that the bacteria in the galls are all intracellular and that they are distributed through the tissues only by the division of diseased cells thus passing some of the germs on to the daughter cells. In this way a group of diseased cells develops and when these push out in a line along the path of least resistance, which is usually parallel to the axis of the plant, a tumor strand is formed as described above.

More recently Riker (14) claims to have demonstrated that the bacteria are intercellular and not intracellular in the diseased tissue. He states that when a needle puncture is made in the plant the tissue for a short distance around the wound becomes water-soaked. The bacteria then migrate, in the water which fills the intercellular spaces, as far as the water-soaked area extends.

In another article (15) Riker discusses some of the responses which the host tissue makes to the organism. He states that "The division of the cells in the early stages of gall formation were observed to form a more or less distinct sheath about the position of the bacteria." Thus if the bacteria should migrate for some distance in a longitudinal direction along a flooded intercellular space, the cells surrounding this space would divide in such manner as to form a sheath along this space, forming a structure very much resembling Smith's "tumor strand".

Nature of crown-gall injury.

During recent years attempts have been made at some experiment stations to determine the exact nature and extent of the damage done by crown gall in orchards. The following extract from a Montana bulletin (10) describes one of these experiments.

"During a time when shipments of nursery stock into Montana were very heavy, the controversy with the nurserymen as to the ultimate effect of crown gall upon apple orchards became so strong that it was decided to set out an orchard of affected and healthy trees and study, year by year, the effect of the disease upon the trees.....

"Detailed notes were taken on each tree before it was set in the orchard. Every diseased tree had at least one gall on the crown, most of them had two or three, and a few had four. These varied in size from one to two and one-half inches. As a rule they extended from one-third to one-half of the way around the crown but a few nearly or quite encircled it. Nearly all had more or less of the hairy-root form of the disease, which on some was very abundant. In other words, these trees all showed the disease in a well-developed condition. Practically all forms of the disease were well represented excepting the aerial type. In one respect the trees were not quite representative of crown-gall stock as it is received by orchardists when no restrictions are placed upon the sale of such stock, in that all trees that had developed hairy root at the expense of good, strong anchor roots had been culled out at the nursery. There was not a single tree of the kind we have often seen in commercial shipments in which the whole root system consists of a mass of hairy roots with no large, strong ones. Every tree in this lot had good, strong roots..... .....

"In September, 1912, (after two and one-half years) notes were taken on all the trees. In general, the growth of both the healthy and the diseased trees had been very good; indeed there was no apparent difference between the corresponding varieties in the two blocks in the matter of trunks and tops. Half of the trees were then dug up and the roots examined. For this purpose every other row diagonally was taken. At this time a marked difference was apparent in the root systems of the healthy and diseased trees, those of the former being in almost every case stronger. Reference to the original notes showed that none of the galls excepting a few of the smaller, softer ones had disappeared but that most of them had enlarged considerably.

"During the summer of 1915 blight was severe in the Bitter Root Valley and these young vigorous trees suffered considerable injury. Some were killed outright, while others were so badly injured that practically the whole top had to be

removed. In the spring of 1916 all the killed and badly blighted trees were pulled out and the difference between the root system of the diseased and the healthy trees was most apparent. A horse was used for this work and in most cases he was unable to remove the healthy trees with a single pull but had to pull repeatedly this way and that to break off the roots. On the other hand, very little trouble was experienced with the diseased trees. With a few exceptions the horse pulled them out at a single attempt, sometimes with very little effort indeed.

"In November, 1917, final notes were taken and the last of the trees were removed as it was necessary to use the land for other experimental work. At this time, after a growth of eight seasons, few of the galls had disappeared entirely and most of them were still growing. Some were several inches in diameter. In some cases there was a general enlargement of the diseased crowns to more than twice the circumference of those of any of the healthy trees. Very few of the new galls, however, had been formed either on the crown or on the roots. The hairy-root condition was very little in evidence and in most cases it could scarcely be found. So far as we could tell none of the trees had died as a direct result of crown gall, though the havoc wrought by blight so complicated matters that we can not say positively that crown gall had killed none. No very great difference was apparent between the tops of the diseased and the healthy trees, though a good observer could notice that the healthy ones had made a little better growth. The root systems of the diseased trees, however, were very inferior.

"The dwarfing effect of the crown gall (on the trunks) was quite apparent. In Northwestern Greening the tops of the diseased trees were fully as good as those of the healthy ones. The Wealthy trees were so reduced in number by blight that a fair comparison was hardly possible. In no case was a tree so markedly dwarfed as one sometimes finds, apparently as a result of crown gall, in a bearing orchard. The reduction in size, however, is much more pronounced than that reported by Hedgcock (8) for a similar experiment carried out in Missouri. The average reduction in circumference in the Montana experiment was 1.58 inches. The average reduction in diameter in the Missouri experiment was 0.29 inch. We preferred to measure circumferences rather than diameters because the irregularities in trunks would have made measurements of the diameter rather uncertain.

"The most striking thing brought out by this experiment is the greatly disturbed balance between the tops and the roots of the affected trees. Doubtless this would have been still greater had not those with no strong anchor roots been culled out and discarded before shipment from the nursery. Whether or not the weakened root systems would have resulted in more serious injury to the tops of the crop after the trees came into heavy bearing can only be surmised. If not, it is of no significance unless the trees have a greater tendency to loosen in the ground under the influence of the high winds. In the sheltered location of this orchard there was no such tendency.

"All things considered, it is safe to say that the increased value of the healthy trees over the galled ones at the time they were removed was much more than the original cost of the nursery stock....."- Swingle, D. B. and H. E. Morris. Crown gall in the orchard. Montana Exp. Sta. Bul. 121. 1918.

Green and Melhus (9) in Iowa performed a similar experiment. A few of their conclusions follow:

"In the experiments recorded in this bulletin on the effect of crown gall on apple trees, two of the leading commercial varieties of apples common in Iowa have been employed, namely, Wealthy and Jonathan. The orchard was set in 1912 at Shenandoah, Iowa, on a modified Missouri loess type of soil, using two year old "cut back" trees for planting.....

"The experiment confirms the general idea that Wealthy is more susceptible to crown gall than Jonathan. When apparently normal Wealthy and Jonathan trees were set in this orchard 62 percent of the Wealthy became infected and 13 percent of the Jonathan.

"In this experiment galled trees did not grow nearly as rapidly as normal trees. Those which were galled when set and later recovered made a greater growth than those that did not recover from the galls.

"The data presented show that crown gall greatly retards the growth activities of young apple trees. It is followed by a reduction in the amount of increase in trunk diameter, and in the number of twigs, their length, their thickness and their weight.....

"Large galls seemed to be more injurious than small ones. Small galls on underground portions of the tree did not seem to injure materially the twig growth.

"Fruit spurs develop earlier on the badly galled than on the normal trees.

"Galls on the stock and union seemed to be equally harmful.

"Galls on the secondary roots were less harmful than those occurring on the union or stock."

Control.

Melhus and Maney (12) carried out some experiments looking toward prevention of infection. They report in part as follows:

"The following tentative conclusions seem justifable from the experiments recorded.

"The infection of apple grafts is readily accomplished by dipping the grafts just before planting in a viable bouillion culture of Bacterium tumefaciens. The majority of the galls occur at the union. The stock is less liable to become infected than the scion.

"Apple grafts were apparently equally susceptible to the crown gall organism where the callous was normal, excessive or slight.

"Well made and poorly made grafts showed little difference in the amount of crown gall that developed.

"Using an unusually large amount of string over the union of the grafts leads to girdling and excessive callousing of the trees, which seems to facilitate crown gall infection. Cloth applied over the union as a wrapper, either with or without string, decreases the amount of crown gall.

"Scion wood cut from trees infected with crown gall at the union did not show any increased amount of crown gall.

"Hairy root seedlings, when used as stock, did not transmit hairy root to the scion, but the stock portion of the graft remained infected in the majority of cases.

"Most of the crown gall infection takes place the first year on the grafts, during the formation of the callous, at the union.

"Surface disinfection with formaldehyde (.16 percent), copper sulfate (.25 percent) and mercuric chloride (.1 percent) were seriously injurious to callousing of apple grafts. Fungicides which go into solution slowly, such as lead arsenate and bordeaux mixture, have a much less injurious effect on the callousing process.

"A strong bordeaux mixture (25-25-50) decreases the stand, tends to have a preserving action on the string and reduces the amount of crown gall.

"Resin sticker added to bordeaux mixture increases its toxic action and reduces the stand. The addition of lead arsenate or soaps to bordeaux mixture does not increase its adhesiveness and its fungicidal efficiency.

"More dilute bordeaux mixtures did not reduce the stand and proved nearly as beneficial in reducing crown gall as the stronger mixtures. The use of bordeaux mixture (8-8-50), with or without lead arsenate, reduced the percentage of crown gall about 66 percent over the checks, and nearly 50 percent over the mean percent of crown gall in all the checks in the Wealthy variety."

Official inspection of nursery stock for crown-gall.

Due to the lack of uniformity in the requirements imposed by inspectors in various parts of the country, a committee was appointed in 1923 to formulate a code whereby more satisfactory results might be obtained without unduly harming either the nurseryman or the purchaser of nursery stock. The report of this committee may be found in Phytopathology 14: 172. March, 1924, and is also put out in pamphlet form by the American Association of Nurserymen, Louisiana, Missouri.

Review.

Compare crown gall with fire-blight as to causal organism, parts of plant attacked, symptoms, effect upon the tissues of the host, means of perpetuation, dissemination and infection, range of host plants and control.

References.

(1) Duggar, B. M. Fungous Diseases of Plants, pp. 114-118.

(2) Smith, E. F. Bacterial Diseases of Plants, pp. 413-472.

(3) Hesler & Whetzel - Manual of Fruit Diseases, pp. 108-112.

(4) Barss, H. P. Crown gall, Oregon Experiment Station Biennial Crop Pest and Horticultural Report, 1911-1912, pp. 218-226. 1913.

(5) Smith, E. F. et. al. Crown gall of plants, U. S. D. A. - B. P. I. Bulletin 213. 1911.

(6) Smith, E. F. et. al. Crown gall. U. S. D. A. - B. P. I. Bulletin 255. 1912.

(7) Smith, E. F. et al. Studies on the crown gall of plants; its relation to human cancer. Botanical Gazette 64: 338. October, 1917.

(8) Smith, E. F. Mechanism of tumor growth in crown gall. Journal of Agricultural Research 8: 165-188. January 29, 1917.

(9) Green, Laurenz and I. E. Melhus. The effect of crown gall upon a young apple orchard. Iowa Experiment Station Research Bulletin 50. 1919.

(10) Swingle, D. B. and H. E. Morris. Crown gall injury in the orchard. Montana Experiment Station Bulletin 121. 1918.

(11) Smith, Clayton O. Further proof of the cause and infectiousness of crown gall. Calif. Exp. Sta. Bul. 235. 1912.

(12) Melhus, I. E. and T. J. Maney. A study of the control of crown gall on apple grafts in the nursery. Iowa Exp. Sta. Research Bul. 69. 1921.

(13) Smith, E. F. Bacterial diseases of plants. pp. 413-472.

(14) Riker, A. J. Some relations of the crown-gall organism to its host tissue. Jour. Agr. Res. 25: 119. July 21, 1923.

(15) ----------Some morphological responses of the host tissue to the crown gall organism. Jour. Agr. Res. 26: 425. Dec. 1, 1923.

(16) Stewart, F. C. Recommendations for the improvement of official inspection for crown-gall. Phytopath. 14: 172. Mar., 1924.

BACTERIAL GUMMOSIS OF CHERRY

Caused by Pseudomonas cerasus Griffin.

This is the most serious of all cherry diseases in Oregon and may occasionally attack other stone fruits besides the cherry. Other names applied to this disease are "Cherry Gummosis" and "bacteriosis". The disease is commonly characterized by a copious flow of gum from the cankers, but it should be emphasized that gumming of cherry and other stone-fruit trees may be caused by any kind of an injury to the bark and the mere presence of gum is not a sure indication that this bacterial organism is present.

1. Laboratory.

a. Symptoms.- Pieces of branches and trunks of cherry trees showing typical cankers should be examined carefully and the symptoms described in the notes. Notice the dead sunken bark, the size and shape of various cankers, the callus around old cankers, the exuding gum, and, in some cases, the blighted buds and spurs. Draw typical specimens.

b. Effect upon the tissues of the host.- Examine cankers to determine how deep the injury goes. Does this organism kill the tissues attacked or simply stimulate them to over-growth? In this respect is it like the fire-blight organism or the one causing crown gall?

c. Life history.- Read the references listed below and describe methods of perpetuation, dissemination and infection, as far as these phenomena are known.

d. Notes.- After reading the references, complete the notes begun in the laboratory.

2. Recitation.- Be ready to discuss in class all phases of this gummosis disease. Get information from any source available.

Text discussion.

Barss (1) gives the best description of this disease available. Parts of his article are quoted below:

"In that section of Oregon lying west of the Cascade mountains the greatest drawback to the culture of our most favored varieties of sweet cherries is the disease commonly known as cherry gummosis.....

"The term "gummosis" by itself denotes simply the abnormal development of gummy or mucilaginous substances, resulting in the formation of gum pockets or exudations from various parts of the plant. The tendency to gum-formation of this kind is characteristic of plums, apricots, peaches, cherries and other stone fruits wherever grown, usually as a response to injury, disease or unsuitable conditions of soil, climate, etc. Citrus and other trees are often subject to similar gumming.

We are concerned here, however, with this phenomenon as it appears on the cherry, especially on the sweet varieties in our own state.

"Of the two groups of cherries, the sour cherries or Prunus cerasus group, and the sweet cherries or Prunus avium group, we find that the former are not nearly so susceptible to gum-flow while the latter seem particularly subject to serious attacks wherever they are cultivated, if we can judge by the reports that come from such widely separated points as Australia, Europe and our own Pacific Coast. In Europe careful scientific investigation of cherry gummosis was begun at about the middle of the last century and has been continued by various persons down to the present.....

"In the State of Oregon serious attacks of gumming in the cherry orchards were noticed at least as early as 1853, very early in the history of fruit raising here, and, from the time of the first reports of the State Board of Horticulture, public attention has been called to the importance of the malady with various suggestions as to causes and remedies. It has been mentioned also in various bulletins issued by the Oregon Experiment Station and by the boards of horticulture and experiment stations of the two neighboring Pacific states.

"It is not known to what extent the cherry industry in other parts of the United States suffers from the presence of gummosis. We hear little from it in the drier, eastern portion of this state; but in the moist valleys of Washington and Oregon, west of the Cascades, at least, the trouble now reaches such proportions as to dishearten many cherry growers and discourage other orchardists from planting cherries.....

"Various Causes of Cherry Gummosis.- As has been intimated, the formation and exudation of gum is to be considered as the result of an injured, diseased or otherwise abnormal condition of the tree. It is a symptom only and not the disease itself. The published literature on cherry gummosis brings to light many explanations for the appearance of this phenomenon, some well proved and others that bear the stamp of probability.

"Injuries.- Mechanical injuries, such as bruises, may induce the formation of gum, but the wound usually heals quickly and the gumming ceases. The injection of certain chemicals into cherry trees has repeatedly caused gum-flow, and such insects as borers may produce it; but these causes need not engage our attention....

"Bacteria as Disease Producers.- There is a disease described by Aderhold and Ruhland in Europe called the bacterial blight of the cherry. Its attacks have done great damage to young cherries in the nurseries especially. In its effects on the plant it is said to be hardly distinguishable from those produced by the Rhine disease before mentioned. The bark is killed, cankers are formed, and trunk or branches may be girdled, and often die in full leaf. Gumming usually, though not always, accompanies the disease. A certain species of bacteria have been isolated from the affected plants and by reinoculation the disease has been reproduced.....

"Description of the Disease in Oregon.- Since numerous distinctly different troubles of the cherry may be accompanied by gum-production, it is evident that the term "cherry gummosis" should not be applied to any specific disease. In this state, however, most of the growers use this expression to designate any

disease of the cherry which may be accompanied by gumming. Cherry gummosis, used in this popular sense, appears in what at first glance seems to be a variety of forms. Closer examination and study shows graduations between most of these types such that it is somewhat difficult to distinguish one from another.....

"The More Serious Phases of the Disease.- The condition most dreaded by growers is that in which the trunk and limbs are affected in a more or less general attack. Very great loss arises in this manner among the young orchards and is apparently at its worst the third or fourth year after the trees are set out. There is very often little indication of the presence of the disease until an entire tree, or one or more branches, fail to leaf out in the spring or a sudden wilting down of a part or whole of the tree takes place at almost any time during the active season of the plant. Careful investigation in such cases reveals the fact that the trunk or branches have been previously girdled by the disease. Sometimes this injury seems to occur without gum-flow, but as a rule there is a more or less copious exudation. The amount of gumming seems to bear no exact relation, however, to the extent of injury. Upon cutting into a tree from which a small amount of gum is oozing at a single spot only, especially where this occurs on the upper part of the trunk, one often finds that a very large area of the tree is dead or dying. In many cases, indeed, external examination does not disclose the existence of a diseased condition until it has made considerable progress. In the later stages, however, there is usually no difficulty in detecting the disease on account of the fact that no further growth takes place at the affected region, while the adjacent and still healthy parts add a new layer of wood during the growing season. The dead area then appears flattened, and, the dead bark, since it does not expand, frequently, though not always, splits open. In other words we have the formation of a canker.....

"The More Restricted and Localized Cankers.- In this disease we find, then, certain conditions in which a large part of the tree may be rapidly and often fatally involved in a general attack, and other conditions in which large cankers or dead areas are formed that may girdle trunk or limbs, but the disease does not always appear in such severe forms, and we commonly find small cankers and the affected spots that are more restricted and localized appearing on various parts of the tree. Near the center of such spots one usually discovers the remains of a dead bud or spur. Sometimes such a dead bud or spur will be found at one side of the canker but connected with it by a streak of dead tissue. This association of small cankers with dead buds is not universal, but it is so common that it suggests the possibility that the diseased spot had its beginning in the death of the bud or spur. In such cases there is sometimes an abundance of gum production and sometimes little. Often an alarming quantity of gum exudes from a spot where only a very small amount of tissue is found to be affected. Again, a canker may entirely girdle a branch with very little exudation occurring or none at all.

"The Blighting of Buds and Spurs.- There is a very common phase of our cherry trouble which has generally escaped the notice of the growers or has been passed by as unworthy of much attention. This is the blighting of buds and fruit spurs, generally accompanied by gumming, which is present in practically all cherry orchards to a greater or less extent, but is much worse in orchards where trunks and limbs are badly diseased. Old trees and younger trees seem to be equally affected. This trouble is first noticeable early in the spring when some of the buds, which formed normally in the fall, fail to swell and open when the others

unfold. A drop of gum often appears exuding from the bud or from the affected spurs. Sometimes affected buds unfold, but before the blossoms open, wilt down and dry up. Often, however, spurs come into full leaf and set fruit, only to die a week or two later. As far as our observation goes, blighting of this sort does not usually take place during the summer or fall.....

"Cherry Gum and Its Formation.- From the preceding discussion it is evident that in the different phases of this cherry disease, the exudation of gum is not an ever-present occurrence. Some of the worst attacks are attended with comparatively little gumming while sometimes diseased spots of very slight extent yield large quantities of gum. There are also seasonal variations in the amount of gumming which have usually been considered indications of a corresponding variation in the severity of the disease, a deduction which might not always be true. In order to correct any wrong impressions as to the nature of cherry gum and the conditions under which it is formed, an explanation of these points will be briefly given.

"The investigations by such authorities as Butler, Sorauer, Mikosch, Bijerinck, Prillieux and others..... have established the fact that the cherry gum is formed through the transformation and liquidation of the walls of certain cell tissues. Such tissues are usually formed abnormally by the cambium (the growing layer of the tree) in response to various stimuli produced through the influence of injury, disease, or disturbed physiological conditions. An enzyme is held by many to be responsible for the actual disintegration of the wall substances into gum, though other explanations have been offered. An abundance of water is required in this process and it seems necessary that the tree be in a growing condition in order that tissues susceptible of transformation into gum may be produced.

"From these statements one would expect the attacks of the disease, when spreading in the dormant season of the tree, would not be accompanied by fresh gumming. A large amount of gum-production, also, would not be expected where there existed a reduced supply of moisture. In this light, it would seem probable that a tree growing vigorously and having an abundant supply of moisture even though diseased in only a few small spots, would produce more gum than a badly affected tree which lacked vigor and was exposed to drought. Observation supports these conclusions.....

"Resistant Stocks and Varieties.- The Mazzard Cherry as a Stock..... It is well known, both in this country and abroad, that the Black Mazzard cherry, the wild sweet cherry of Europe, is much hardier and less liable to suffer from adverse conditions than are the cultivated varieties. Hence, seedling Mazzards have come to be much used in Europe as stocks upon which to graft the commercial sweet varieties. In this country also the Mazzard is coming to be recognized as a sturdy stock which unites with the sweet cherries better than the Mahaleb. A point in favor of the Mazzard as a stock is that it seldom gums.....

".....The writer is convinced that the use of the Mazzard as a stock on which to limb-graft or bud the other varieties, is a thoroughly practical way of protecting the cherry from this disease in its most damaging form.....

"Cutting out the Disease.- Whatever may be the primary cause of cherry gummosis, the spreading of the injuries is probably due to the presence in the affected parts, of organisms that work out into healthy tissues and cause their death. Be this as it may, the most successful method of treatment in practice among careful cherry raisers consists in the thorough cutting-out early in the season of all diseased, discolored and gum-soaked bark. Persistent watch is kept by frequent inspections and as soon as new diseased spots are discovered, they are cut out. Where this has been conscientiously done, and the injuries have been cut out before getting very large, the recoveries have been rapid and the damage from the disease rather small, as far as the writer's observations have gone. In cutting out it is important that all the tissues which are in any degree affected should be removed..... It is often impossible to discover at once all the affected tissue around a gum exudation or canker, and a second or third cutting-out may have to be made during a season, but persistence will be rewarded in most cases by a rapid healing of the wound through the growth of new callus tissue over it. Care should be taken not to remove any more of the living and healthy tissues than is necessary in cutting out the disease.....

"Where a small branch is affected, it is often better to remove it entirely and allow a healthy one to take its place. In young trees it is strongly recommended to remove all blighted spurs and cut away discolored tissues that spread out from their base; since our investigations lead us to suspect that many of the serious cankers originate in this way.....

"Spraying seems to be practically useless as a means of control for cherry gummosis."- Barss, H. P. Cherry Gummosis. A Preliminary Report. Oregon Agricultural College Biennial Crop Pest and Horticultural Report. pp. 199-216. 1911-12.

In a later article Barss (2) gives the results of further experimental work with this disease. His summary of the main points brought out by the later investigations follows:

"1. It has been proved by a large number of successful isolations, inoculations, re-isolations and re-inoculations that the most serious disease of sweet cherries occurring in Western Oregon is caused by bacteria.....

"2. The disease in nature causes a blighting of buds and spurs and the forming of cankers on branches, limbs and trunks which often result in the death of the parts attacked. These effects have all been reproduced by artificial inoculations.

"3. The exact manner in which the disease is disseminated is not known, but indications seem to point to the possibility that sucking insects may be largely responsible for infections.

"4. The period of most serious infection is believed to be the autumn. The new infection appears to develop slowly at first, gradually gathering momentum and reaching its maximum rate of advance in the late winter or early spring.

"5. The progress of the cankers usually receives a sudden check through the formation early in the spring by the tree of a wound-cork barrier followed by callus formation. Cankers seldom or perhaps never spread after the first season.

"6. Wound parasites and wood-rotting fungi frequently enter at the cankered spots and extend the injury originally caused by the bacteria.

"7. Observations point to the general resistance of the so-called Mazzard seedlings to the disease. Among commercial varieties, the Lambert is more resistant than either the Royal Ann or Bing.

"8. A similar canker disease has been found on prunes, peaches and apricots, and pathogenic bacteria apparently identical with the cherry organism have been isolated. The effects on other stone fruits seem to be in general less serious than on the cherry.

"9. It is suggested as a means of prevention that resistant seedlings be planted and limb-grafted to the commercial varieties later. Surgical treatment is recommended for control of the disease in the orchard and the wrapping of trunks and limbs for the prevention of infections is a method proposed for trial."- Barss, H. P. Oregon Agricultural College Biennial Crop Pest and Horticultural Report. 1913-14. pp. 239-240.

Review

Compare bacterial gummosis of cherry with the bacterial diseases previously studied in all details. At what season of the year is the disease active? When dormant? Discuss control measures.

References.

(1) Barss, H. P. Cherry Gummosis, a preliminary report. Oregon Experiment Station Crop Pest and Horticultural Report. 1911-12. page 199. 1913.

(2) Barss, H. P. Bacterial Gummosis or Bacterial Canker of Cherries. Oregon Experiment Station Crop Pest and Horticultural Report 1913-14, page 224. 1915.

(3) Barss, H. P. Bacterial Gummosis of Stone Fruits. Monthly Bulletin of the California Commission of Horticulture. March, 1918.

(4) Hesler and Whetzel. Manual of Fruit Diseases, page 181.

WALNUT BLIGHT

Caused by Pseudomonas juglandis Pierce

This disease has long been known in California where the English or Persian walnut is extensively grown. Some English walnuts are grown in Oregon also and the blight has been known in this state for a number of years. It has been reported also from the eastern part of the United States.

The disease frequently does considerable damage, especially when it attacks the fruits.

1. Laboratory.

a. Symptoms.- Examine diseased specimens of twigs, leaves and fruit. Make sketches to show the effect of the disease upon these parts and describe in your notes.

Text discussion.

Hosts and distribution are discussed by Smith, Smith and Ramsey (2) as follows:

"This is by far the most important trouble affecting the walnut in California. So serious has been this disease that the loss of a large portion of the crop has in some years been charged to this source; legislatures have made special appropriations for its investigation, and the growers have offered a large reward for a practical remedy.

"Host.- The natural occurrence of bacteriosis or walnut blight is probably confined to the English walnut, or its hybrids with the blacks. Blighted nuts have been collected from a Paradox walnut tree (a hybrid of English and California black) and the organism has also been isolated from the leaves of Paradox seedlings growing in the Experiment Station nursery. The disease has never been found on the straight California black or on Royal hybrids, although it can be produced artificially by puncture inoculation on various species of Juglans.

"Geographical Distribution.- It is not definitely known just how widely spread this disease may be. In the United States it is found more or less scattered over California, some localities being more severely infected than others, due either to climatic conditions or to the disease not yet having obtained a foothold. California has had the disease for a quarter of a century, and now it is beginning to show itself in Oregon. The disease has also been reported from Texas, and is said there to attack nearly every year all the native nuts of the walnut family. This Texas trouble has not yet been with certainty identified as the same disease as the walnut bacteriosis in California. Diseased walnut leaves have been received from at least one point about midway down the Pacific coast of Mexico. From this diseased material typical walnut blight cultures were grown. The walnut trees were, however, imported from California. This disease will probably appear

wherever the English walnut can be commercially grown in the United States, and the only reason that it is of no economic importance in the Southern and Eastern States is because the walnut industry has not yet been developed there. In foreign countries the disease has been reported from New Zealand. California walnut growers who have visited France report having seen our trouble on French walnut trees."- Smith, R. E., Clayton O. Smith and Henry J. Ramsey. California Experiment Station Bulletin 231. 1912.

McMurran (3) gives additional information on distribution in the eastern half of the United States.

"Walnut blight, or bacteriosis, is distributed very generally throughout the eastern half of the country. Investigations by Mr. M. B. Waite and the writer have demonstrated its occurrence in Louisiana, the District of Columbia, Maryland, Delaware, Pennsylvania, and New York, and there seems to be no reason to suppose that it will not occur wherever Persian walnuts are grown in the United States."- McMurran, S. M. Walnut blight in the eastern United States. U. S. Dept. of Agr. Bul. 611. Dec. 10, 1917.

Further information on losses, symptoms and life history is given by Smith, Smith and Ramsey (2) and on control by Smith and Smith (1).

"Losses from Blight.- The amount of loss actually caused by this disease has been every year very large for the past ten years or more, but has varied considerable from year to year. It is also true that much has been attributed to blight which in reality had nothing to do with this disease, such as the effects of drouth, frost and other unfavorable climatic or soil conditions. At the same time the losses directly attributable to blight have been extremely large. One significant fact in this connection is that while the walnut acreage in southern California has multiplied many times during the past decade, the total walnut crop of the State has increased very little during this time. In fact, the crop has been actually much less during several years of this period than it was in some of the best years of the preceding decade. This loss or failure of the crop to increase has not been entirely attributable to blight, yet it has certainly been due to the disease much more than to any other one factor. In the year 1903, one of the first bad blight years, the total crop of the State fell off to only a little more than one half of that of the previous year. It is probably conservative to state that in the seedling groves of southern California an average loss of at least 50 per cent of the crop which would otherwise have been harvested has been caused by the blight during the past ten years.

"Cause of Disease.- The disease thus far discussed has been conclusively proven to be produced by a species of bacteria growing in the diseased parts, and hence is very properly referred to as Bacteriosis. A microscopic examination of diseased tissue shows countless numbers of these small, rod-shaped organisms to be present. By employing bacteriological methods pure cultures were used in making artificial inoculations into healthy nuts and shoots, thus again producing the typical disease. The organism has been isolated many times, and no difficulty has been experienced in always producing the disease by inoculation from culture if the tissue was in an active growing condition.

"Characteristics of Bacteriosis.- Walnut bacteriosis may occur on all the tender, new, growing parts of the tree, such as young nuts and branches, the petioles of leaves, as well as mid-veins, the fine lateral veins and adjoining parenchyma. On the affected parts characteristic blackish colored areas or quite pronounced lesions are produced. The blackening is, as Pierce suggests, brought about by oxidation of the tannic acid in the tissue. At any point of injury in the tender growth of the walnut a very similar black coloration will occur.

"On Branches.- Bacteriosis is at first confined to small areas, but under favorable conditions these increase in size to a lesion or diseased area extending two or three inches in length on the green shoot. The disease always has its beginning on the young succulent growth which may be near the growing end or at any other point. When the disease infects a branch near its end, that part may be killed back, but this seldom occurs except when the diseased lesion is very near the end. In the worst diseased lesions the tissue is killed inwardly to the pith, while in less severe cases only the bark and wood are diseased. As the shoot becomes more woody it is more and more difficult to infect and no tissue ever becomes affected after the first few months of its growth. The disease after the first year, even in well define lesions, gradually dies out and the tissue heals over the old lesion, although in some cases short lengths of the worst diseased shoots may die back for a few inches. The diseased portion on the twig at first forms a small discolored or water-soaked area which gradually increases in size, and at length the central portion is black and is surrounded by a water-soaked margin or fermentation zone. As the shoot becomes more and more woody the active development of the disease is checked and no further tissue is involved. Then the whole diseased area becomes blackened in color. The diseased portion in many cases comes to have a somewhat shrunken, dried-out, deformed, cracked condition because of the killing and drying-out of the tissue.

"Leaves.- The leaves are sometimes diseased, especially the petioles and veins, which become a blackish or brownish color. The disease also attacks the parenchyma or soft tissue of the leaf, causing spots of a brownish color and of various shapes. These spots are rather common on rapidly-growing leaves, and especially on the very large leaflets of young nursery trees. The spots are circular or angular in outline and by confluence often cause quite large diseased areas. Here the small lateral veins in the diseased areas are a browner color than healthy ones. The disease does not cause serious defoliation of the tree and should not be confused with the falling of the leaves that sometimes takes place after a period of hot weather during the summer months.

"Nuts.- Bacteriosis on the young nuts is especially virulent and destructive under favorable conditions. The disease on the twigs and leaves ordinarily does very little injury and the disease would be of little economic importance did it not attack the nuts.

"Blossom End Infection.- While the nuts may be infected at any point in the surface, by far the most common as well as most virulent form of infection is at the blossom end near what is technically called the stigma. This is the weakest part of the nut and is especially sensitive to the blight. The bacteriosis is very bad on the small nuts, and when once it has started at this point it rapidly continues its growth within the tissue, until the small nut is sufficiently weakened to fall.

"Lateral Nut Infection.- The disease on the nuts starts at one or more points of the surface as a small, circular, raised, discolored area that at first has a water-soaked appearance and may not be larger than the point or head of a pin when first visible. The diseased area in its earliest stages is slightly raised above the surrounding healthy tissue, but as the disease progresses, the spot becomes more or less sunken below the normal tissue. The spot gradually enlarges in size and becomes black in appearance. Surrounding the blackened area is a paler zone, having the same water-soaked appearance already referred to. This band of tissue lies between the healthy tissue and the blackened area and represents the cells of the tissue that are being acted upon by the ferments secreted by the organism, which break down the tissue and prepare the way for the further advance of the bacteria. In the early infection, if climatic conditions are favorable, lesions or dark spots are formed which often extend through the hull and shell-forming tissues into the kernel. The nut in such cases is deformed in shape as the diseased part ceases to grow. Such nuts do not bark clean, as the outer covering clings very tightly to the shell, and the kernel at best is only poorly developed.

"Winter Habitat of Germ.- The germ of the organism without question winters in the old lesions of the branches. Much work has been done in making cultures at short intervals of time throughout the year from the different diseased tissues in order to see if the disease organism was alive and where it could best pass through the winter or dormant period. In every series of cultures the disease organism was found, showing conclusively that the disease was still alive in the old lesions of the wood and bark. The cultures made from the old nuts, both those on the ground and some still on the tree, gave some walnut blight cultures early in the autumn, but as the season advanced the organism seemed to die out and toward spring it was almost impossible to secure blight cultures from the old nuts found about the orchard.

"How the Germ Enters the Tissue.- Nuts, as well as the leaves and young shoots, are provided with stomata or breathing pores through the epidermis into the interior of the tissue. These stomata on the nuts are arranged in groups of from five to a dozen or more, and are to be found on the paler green specks that spot the surface of the husk of the green nuts. It is through these openings that the bacterial organism can gain entrance to the interior of the tissue of the young nuts."- Smith, Ralph E., Clayton O. Smith, and Henry J. Ramsey. Walnut Culture in California: Walnut Blight. California Experiment Station Bulletin 231. August, 1912.

Control.- "No practical remedy has been found for this, but the problem has been largely solved by the development of immune varieties. Such trees are now available to a limited extent and must soon supplant the present plantings of seedlings or susceptible kinds.

"Some control may possibly be obtained by Spraying with Bordeaux mixture or sulfur sprays just before the buds open. The expense of spraying walnut trees thoroughly is so great, and so much time is required to thoroughly cover trees of this size, that we have no faith in a satisfactory solution of the walnut blight problem along this line.

"On old groves liberal fertilization with nitrogenous fertilizer, together with the use of green manure crops, will go far towards making up for the losses

caused by the disease."- Smith, R. E. and Elizabeth H. Smith. California Experiment Station Bulletin 218. June, 1911. pp. 1172-73.

Review.

In what ways does walnut blight resemble fire blight and in what ways does it differ from fire blight? What phase of the disease seems to do most damage? Discuss the difficulties in the way of satisfactory control of this disease. Does walnut blight differ from other bacterial diseases studied in difficulty of dontrol?

References.

(1) Smith, R. E. and Elizabeth H. Smith. Walnut blight or bacteriosis. California Experiment Station Bulletin 218, p. 1172. June 1911.

(2) Smith, R. E., C. O. Smith and H. J. Ramsey. Walnut blight. California Experiment Station Bulletin 231, page 320. August, 1912.

(3) McMurran, S. M. Walnut Blight in the Eastern United States. U. S. D. A. Bulletin 611. December 10, 1917.

(4) Barss, H. P. Walnut diseases. Oregon Experiment Station Biennial Crop Pest and Horticultural Report, 1911-12. Page 260. 1913.

CITRUS CANKER.

Caused by Pseudomonas citri Hasse.

Citrus canker is a good example of a foreign disease which, when introduced into the United States caused consternation among citrus growers and required heroic measures to control it. The following quoted discussion (1) gives the main facts about this disease in brief form:

"Historical

"Citrus canker was introduced into the Gulf States from Japan on nursery stock. It has been found to occur in Japan, the Philippine Islands and parts of Eastern Asia. Whether or not it is indigenous to these countries is not known with certainty, but it is known that it is not of American origin. Specimens were first brought to the writer's attention in February, 1914. The Office of Nursery Inspection of Florida had collected specimens in September, 1912, but did not realize the presence of a new citrus disease within the State until July of the following

year. The disease has been collected in all of the Gulf States in the sections adapted to the growth of citrus trees. Within Alabama it is confined principally to Baldwin and Mobile counties. A very small percentage of the trees within the state are affected with citrus canker, but when the infectious and virulent nature of the disease is realized it is seen that these few trees are a serious menace to the citrus industry in Alabama. It is for this reason that such energetic efforts are being made to prevent its further introduction and spread and to eradicate the disease already present.

"Hosts.

"Citrus canker has been found to occur on many of the species and varieties of citrus. Among them are grape-fruit, King oranges, mandarines, Satsumas, tangerines, lemons and limes. It has been observed in Louisiana on species of Fortunella, to which genus the kumquat oranges belong. Swingle has recorded the presence of canker on kumquats in Japan....."- F. A. Wolf, Citrus Canker. Ala. Agr. Exp. Sta. Bul. 190. May, 1916.

The following items on appearance, cause and control are quoted from Stevens (3).

"Appearance.

"The distinguishing feature of citrus canker as observed in the field is the characteristic spotting produced on the fruit and foliage. As usually seen, the infection appears as small, light brown spots, from less than one-sixteenth to one-quarter of an inch in diameter. The spots are usually round and may occur singly, or several may run together, forming an irregular area. This last condition usually occurs on fruits. The spots are raised above the surrounding healthy tissue, and are composed of a spongy mass of dead cells, covered by a thin white or grayish membrane. The membrane finally ruptures and turns outward forming a lacerated or ragged margin around the spot.

"On the leaves, infections first appear as small, watery dots, with raised convex surfaces. These dots are usually of a darker green than the surrounding tissue. Sometimes, however, the surface of the spots is broken as soon as they appear. Spots may appear on either surface of the leaf, but they do not at first penetrate thru the leaf tissue. They gradually increase in size, change to a light brown and become visible on both sides of the leaf. In the older spots one or both surfaces may be bulged or raised, and such spots are commonly surrounded by a narrow, yellowish band or zone. In the more advanced stages the surface of the spots becomes white or grayish, and finally ruptures, exposing a light brown spongy central mass. Old spots soon become overgrown by saprophytic fungi, and may appear pink or black on account of these fungus growths.

"On the fruits the spots are very similar to those formed on the leaves. They project, and retain a circular outline. They do not penetrate far into the rind. They may be scattered over the surface, or several may occur together forming an irregular mass. Gumming is sometimes associated with the spots formed on the fruits. Canker apparently does not cause a rot of the fruits directly but opens the way for other fungi to enter and cause infected fruits to rot.

"The spots on young citrus twigs are like those on the leaves and fruit. On the older twigs they are more prominent, and more or less irregular in shape. This is especially true of old spots. They show the same spongy tissue as is found in the spots on the leaves, but assume a cankerous appearance and the surface membrane completely disappears. These spots or cankers are formed in the center of the outer layers of the bark tissue, and do not penetrate into or kill the wood. The spots once formed in the bark are persistant and are not readily sloughed off. They may remain for a long time, and form centers from which infections may readily spread.....

Cause.

"Citrus canker is a bacterial disease. It is caused by Pseudomonas citri, a short rod shaped, motile organism that can be seen only with a compound microscope. These bacteria are found in countless numbers in the canker spots and they are exuded in masses whenever the spots become moistened by rain or dew. Drops of water contaminated with bacteria spread the disease and when the organisms come in contract with citrus tissue, especially the tender foliage, twigs and fruit, new canker spots are rapidly formed. In the older bark on the branches and trunks of trees the bacteria probably gain entrance thru small wounds or cracks.

"The disease may be spread some distance by the wind and beating rains, or any agent coming in contact with the moistened foliage of an infected tree is apt to spread the disease to other trees. The bacteria are known to live and multiply in moist soil and they are capable of surviving in the soil for many months and still retain their virulent nature. Soil infected with the canker bacteria has no doubt been an important source in spreading the disease in many cases."

Control.

"The prompt and complete destruction of all canker infected trees is the only practical method that has yet been found for checking the disease in Florida."

References.

(1) Wolf, F. A. Citrus Canker. Alabama Exp. Sta. Bul. 190. 1916.

(2) Wolf, F. A. Citrus Canker, Jour. Agr. Res. 6: 69. 1916.

(3) Stevens, H. E. Florida Citrus diseases, Florida Exp. Sta. Bul. 150. pp. 62-68. 1918.

(4) Peltier, Geo. L. and David C. Neal. Overwintering of the citrus-canker organism in the bark tissue of hardy citrus hybrids. Jour. Agr. Res. 14: 523. Sept. 9, 1918.

(5) Peltier, Geo. L. and Wm. J. Frederick. Relative susceptibility to citrus-canker of different species and hybrids of the genus Citrus, including the wild relatives. Jour. Agr. Res. 19: 339. July 15, 1920.

BLACKLEG OF POTATO

Caused by Bacillus phytophthorus Appel.

This disease or closely related forms of it has a very wide distribution, occurring in many states of the Union, including Oregon, as well as in various countries outside the United States. It is not one of the worst potato diseases but may become serious at times.

1. Laboratory Study.

Examine samples of diseased plants showing effect on stems and tubers. Sketch.

Read the following discussion or some of the references appended and write up the notes.

2. Text discussion.

The following quotations from Morse (1), and from Rosenbaum and Ramsey (2) give a brief account of this disease.

"Blackleg is a bacterial disease of the stem and tuber of the potato. A similar appearing malady caused by bacteria has been reported from Canada, and another from England, Germany, France and other parts of Europe. Preliminary studies of the organisms associated with the disease indicate that they are closely related to those already described as a cause of similar troubles elsewhere, but whether they are identical with any of the described species of bacteria is not fully determined.

"The attacked plants are usually unthrifty, light green or even yellow, and undersized. The branches and leaves have a tendency to grow upward forming a rather compact top, often with the young leaves curled and folded up along the mid-rib. The most characteristic thing about them is the inky-black discoloration of the stem, at or below the surface of the ground, but frequently running up the stem from one to several inches above ground. The seed-piece from which the attacked plants spring is invariably attacked with a soft-rot, and the disease appears to start on the stem at its junction with the diseased seed tuber. The germs of the disease are capable of causing a rapid decay of the young tubers, and these are sometimes attacked also.

"The evidence thus far obtained indicates that blackleg is largely distributed by means of germs carried in wounds, cracks and decayed areas of seed tubers. On account of the readiness with which the organisms are killed by drying there is little to fear from sound, smooth seed stock, but this should be treated with a disinfecting solution as a matter of precaution. There is some reason to think that blackleg was introduced into Canada from England and from there to the United States.

"Blackleg is apparently becoming quite widely distributed throughout the Eastern part of the United States. In most states it is not common enough to attract attention, and in no region has it done much damage, although it may become a serious pest in some sections.....

"The propogation and spread of the disease probably can be controlled largely by the selection of seed from fields free from the disease, the rejection of all seed tubers which have wounds, cracks or decayed areas and treating the remainder with corrosive sublimate..... It is not known whether or not the disease germs will remain alive in the soil to infect future crops of potatoes, but as a precautionary measure the land on which the disease occurs should be kept in grass, clover or cereals for as long a time as possible before planting it to potatoes again."--Morse, W. J. Blackleg: a bacterial disease of the Irish potato. Maine Exp. Sta. Bul. 174. 1909.

Effect of weather.

"The severity of the disease during the growing season is closely correlated with temperature and precipitation and is dependent upon them. A high temperature and low precipitation tend to diminish the disease, while a low temperature and high precipitation produce conditions favorable for it....."- Rosenbaum, J., and G. B. Ramsey. Influence of Temperature and precipitation on the blackleg of potato. Jour. Agr. Res. 13:507. June 3, 1918.

References.

(1) Morse, W. J. Blackleg: a bacterial disease of the Irish potato. Maine Exp. Sta. Bul. 174. 1909.

(2) _____ Control of blackleg disease of the potato. Maine Exp. Sta. Bul. 194. 1911.

(3) Rosenbaum, J. and G. B. Ramsey. Influence of temperature and precipitation on the blackleg of potato. Jour. Agr. Res. 13: 507. June 3, 1918.

(4) Artschwager, Ernst. Pathological anatomy of potato blackleg. Jour. Agr. Res. 20: 325. Nov. 15, 1920.

BLACK-ROT OF CRUCIFERS.

Caused by Bacterium campestre (Pammel) E F S

This disease is found in both Europe and America. In some localities at times it may do considerable damage.

1. Laboratory Study.

Note the symptoms on leaves and stems. Cut the stem and note effect on vascular system. Draw.

After reading the following or some of the references cited, write up a brief account of this disease.

2. Text discussion.

Stewart and Harding (2) give the following description of the symptoms and mode of infection of this disease:

"Appearance of affected plants.

"The first evidence of disease usually appears in the latter part of July when the more advanced plants of late cabbage are beginning to form heads. The first symptoms of an outbreak are easily recognized on a hot, dry afternoon when a number of the plants will appear wilted and lighter green in color. A cross section of the stem of these plants near the ground shows that many of the water-carrying fibro-vascular bundles are black; and on splitting the stem these black lines can be followed down to the withered extremity of the tap root. A diseased condition of any considerable number of these bundles curtails the water supply and when atmospheric conditions are favorable for rapid evaporation from the leaves the latter quickly wilt.

"The upward movement of the water carries the disease along the bundles out into the leaves. As soon as the bundles supplying any considerable portion of a leaf become diseased that part of the leaf dies for lack of water. The blade of the leaf becomes light brown and has a texture like parchment. It is semi-transparent and when closely examined the network of fine veinlets which have been turned black by the disease stands out sharply in the brown background.

Early in August the disease commonly manifests itself in another form. Brown spots appear at the margin of many of the leaves, especially of those which come in contact with the soil. These brown areas spread toward the center of the leaf and a close examination shows the fine veins to be blackened. In from one to three weeks, depending on circumstances, the disease usually reaches the stem of the plant. The progress of the disease from this point is identical with that brought about by infection through the root.

"In either form of infection the most reliable diagnostic character of the disease is the blackening of the fibro-vascular bundles. These bundles may be readily inspected by cutting across the leaf petiole or the stem.

"The failure to supply sufficient water checks growth and often results in the death of the plant. The fibro-vascular bundles do not branch freely in the stem and in cases where the disease gains a foothold only on one side of the plant the growth on that side is retarded so as to produce a marked curvature. The lower leaves turn brown and drop off, but when the plant succeeds in forming a head the upper leaves are held in place and often turn black and decay thus destroying the commercial value of the head....."

"Mode of Infection.

"There are at least three avenues thru which these germs may gain access to the plants:

(1) At the time of transplanting into the field some of the roots are broken, exposing the ends of the fibro-vascular bundles to the attack of P. campestris in the soil. The plants which wilt badly within a few weeks often have many black fibro-vascular bundles in the stem when there is no evidence of the disease in the leaves. This mode of infection seems to be most active during the early life of the plants.

(2) Insects, by eating the leaf tissue, expose the cut ends of the fibro-vascular bundles and either infect these directly by their jaws or leave the surfaces in condition to be infected from other sources. This avenue of infection appears to be more important with cauliflower than with cabbage, but in either case is of secondary importance.

(3) Under favorable atmospheric conditions the water pores at the margin of the leaves exude liquid. Any germs which find their way into these drops after swimming back thru the opening of the water pore find themselves at the termination of a fibro-vascular bundle. A great majority of the infections occurring during the month of August can be traced to this source. Sometimes as many as a hundred cases of this form of infection may be seen on a single large plant."- Stewart, F. C., and H. A. Harding. Combating the black rot of cabbage by the removal of affected leaves. N. Y. (Geneva) Agr. Exp. Sta. Bul. 232. April, 1903.

Control

Edgerton (1) is authority for the following control measures:

"Seed Treatment.- Seeds of all plants liable to be injured by the black rot, including cabbages, cauliflowers, collards, turnips and other related plants, should be treated in a solution of corrosive sublimate. This material can be obtained at any drug store in the form of tablets. Following the directions given on the container, a 1-1000 solution should be prepared. This usually means one tablet dissolved in a pint of water. The solution should be prepared in a glass,

wooden or earthenware vessel, never in a metallic vessel. The seed are soaked in this solution for thirty minutes, then thoroughly washed in clean water and allowed to dry. They should be planted as soon after the treatment as possible.....

"Seed bed sanitation.- Seed beds are necessary evils. There is no satisfactory way for the trucker to grow his young plants except in seed beds, yet many diseases thrive in the seed beds and are later taken to the fields. The black rot is one of the diseases that often obtains a foothold in the seed bed and is afterwards carried to the field on the young plants. Only by efficient sanitary measures can this be prevented. The measures which should be adopted in order to keep diseases out of the seed beds include the following:

1. The seed beds should be placed on high ground and removed from fields that have previously grown the crop.

2. Nothing should be placed in the seed beds that has been in contact with infectious material. Often infected plants from the packing sheds find their way to the manure heap and then to the new seed beds. No manure should be used unless it is known to be absolutely free from infection.

3. For the seed beds, soil from the woods or new ground is preferable. If such is not available, it should be obtained from fields that have not grown the crops which are to be planted.

4. Unless there is some good reason for not doing so, it is best to change the seed bed to a new locality each year.

5. The young plants in the seed-bed should have plenty of light and ventilation and should at all times be in a healthy and resistant condition."

"Rotation.- As many diseases live over from one season to the next either in the soil or on dead plants that are left in the field, rotation of crops is essential. On account of the black rot and another disease known as the Sclerotinia rot, cabbages should not follow cabbages, cauliflowers, collards, turnips and related plants or crops like lettuce, endive and escarole.

"Destruction of Diseased Plants.- At the end of the season, it is a good plan to pull and pile all the old plants and after they are dry, burn them....."

References.

(1) Edgerton, C. W. The black-rot and other diseases of cabbage. Louisiana Extension Cir. 64. 1923.

(2) Stewart, F. C., and H. A. Harding. Combating the black-rot of cabbage by the removal of affected leaves. N.Y. (Geneva) Exp. Sta. Bul. 232. 1903.

(3) Harter, L. L. and L. R. Jones. Cabbage diseases. U. S. D. A. Farmers' Bul. 925. 1918.

(4) Walker, J. C. and W. B. Tisdale. Observations on the seed transmission of the cabbage black rot organism. Phytopathology 10: 174. Mar., 1920.

BACTERIAL WILT OF CUCURBITS.

Caused by Bacillus tracheiphilus E F S

The following brief quotations from Duggar (1) and from Rand and Enlows (2) give the main facts concerning this disease. Further information may be found in the appended references.

"Symptoms.- The general symptoms are simple and striking. These consist of a progressive wilting of the host. If infection takes place upon the central stem, the wilting in the whole vine follows promptly. If, however, infection is in the distal parts of the remaining branches, there is gradual wilting back to the main stem. Then the remaining branches promptly show the effect. In the tissues there is at the time of wilting very slight, if any, evidence of a change in appearance. In no case is there the development of odors, or of decay in the usual sense.

"Infection and spread of the disease appears to result almost wholly through biting insects. The organism is found massed primarily in the vessels of the xylem. At first the spiral vessels are the seat of action, and later the pitted vessels are infested. In late stages of the disease the lesions may be considerable, the bundle system being broken down and cavities formed in the adjacent tissues. The lesions are also very noticeable when the organism has gained entrance to the fruit....."- Duggar, B. M. Fungous Diseases of Plants. 1909. pp. 129-130.

Distribution and hosts.

"The disease occurs in 31 States, including the territory from Vermont and Canada to Florida and west to Minnesota, Nebraska, Colorado, and Texas. It also probably occurs in parts of California. Of our common domestic cucurbits the disease affects cucumbers, cantaloupes, summer and winter squashes, and pumpkins, but not watermelons.

Damage.

"The severity of the disease varies widely in different seasons and localities from an occasional wilted plant up to a destruction of 75 to 95 per cent of the crop. There is very little direct relation between percentage of infections and severity of the disease and ordinary weather conditions in the North, but there is a direct relation to prevalence of cucumber beetles and condition of vigor in the host plant.....

Life history.

"It has been definitely proved that bacterial wilt of cucurbitis does not winter over in the soil, and all seed tests have given negative results. The disease has been carried experimentally for six weeks by striped cucumber beetles

hibernated artificially in cold storage. We have thus far been unable to hibernate the beetles for a longer period.

"Considerable circumstantial evidence, however, points to cucumber beetles as winter carriers. For example, among the first collections of beetles in the spring a small percentage were found to be wilt carriers. The earliest cucurbits were still very small and no wilt was anywhere in evidence. Furthermore, a careful record of spring sequence of wilt was kept in all field and garden plats of cucurbits in two localities and in all cases the leaves first showing wilt had plainly started from these injured points, and there was a tendency for wilt to spread in groups around the original cases, in each new plant starting from beetle-injured leaves. In many of the cases such beetle injuries had not become general at this time throughout the fields.....

Control.

"Where the disease is likely to be severe, spraying with strong Bordeaux mixture and lead arsenate powder (4-5-50-2) is recommended. Treatments should begin as soon as the cucumber plants develop their first true leaves and should continue at intervals of about a week until the cucumber beetles practically disappear from the field.....

"The pulling of wilted vines during the first part of the season, or as long as it can be done without mechanically injuring the healthy plants, will greatly assist in controlling bacterial wilt if consistently done in all neighboring fields. The diseased vines should be buried or otherwise removed from access by the beetles.....

"For control in greenhouses the beetles should be kept out in the first place if possible. Do not grow cucurbits nor pile cucurbit refuse in the immediate vicinity of greenhouses, as this attracts the beetles and many will later find their way into the houses. If the beetles once gain entrance to a house filled with growing plants hand picking is the only remedy to be recommended until some fumigant is found that will kill the beetles without injuring the cucumber plants. Besides destroying the cucumber beetles, great care must be exercised in disinfecting all instruments used in pruning wilted vines before using them again on healthy plants. This may easily be done with a bottle of 1 to 1,000 mercuric chloride and a sponge....."- Rand, Frederick V., and Ella M. A. Enlows. Bacterial wilt of cucurbitis, U. S. D. A. Bul. No. 828. May 10, 1920. pp. 41-43.

References

(1) Duggar, B. M. Fungous Diseases of Plants, 1909. pp. 129-131.

(2) Rand, Frederick V., and Ella M. A. Emlows. Bacterial wilt of cucurbits U. S. D. A. Bul. 828. 1920.

(3) _____ Transmission and control of bacterial wilt of cucurbits. Jour. Agr. Res. 6: 417. 1916.

BEAN BLIGHT

Caused by Bacterium phaseoli E. F. Smith.

This disease is of widespread occurrence and frequently does serious damage. The following articles will give one a good idea of the symptoms of the disease and other useful information concerning the spread of the disease and its control.

Below are extracts from Michigan and Oklahoma bulletins giving the main facts concerning this important bean disease.

History.- "So far as is known the disease is of American origin. Its presence was first reported by Beach, (4) New York, in 1892. The same year Halstead, New Jersey, described the disease and stated that it had been known in that state as early as 1886. These workers described the effects of the disease upon the plants, and as a result of microscopic examinations ascribed the cause to bacteria. Dr. E. F. Smith, U.S.D.A. in 1897 isolated, named and briefly described the organism. Delacroix in 1896 described the same or a very similar disease, occurring on varieties of beans grown on the outskirts of Paris.

"In 1901, Dr. E. F. Smith, U.S.D.A., published a brief account of the growth of this organism on certain media. Halstead, New Jersey, in the same year briefly described the field characters of this disease and experimented with irrigation, shading, sprinkling, old and new land and ridging as control measures. He also mentions varietal disease resistance. Sackett, of the Michigan Station, in 1905 published a very brief account of the disease, and in 1906 Whetzel, New York, published a similar work. Little new data on control was embodied in these papers. In 1908 Fulton, of Louisiana, described the field characteristics of the disease and took up the matter of prevention and of control by seed treatment with hot water, spraying and disease resistant varieties. Edgerton and Moreland, also of the Louisiana Station, in 1913 presented further data on bacterial blight. Their work showed the bacteria markedly resistant to dying. Inoculation experiments were successful. They recommended the use of home grown seed and tried seed treatment with hot water and various chemicals. Muncie, of Michigan, in 1914 and 1917 describes the disease in the field, and discusses its relation to weather conditions and the mode of dissemination. He proves that the bacteria survive the winter in the soil and on diseased bean trash. Control with chemical solutions, and wet and dry heat were tried and resulted in failure. Spraying was tried. The use of clean seed of western grown Michigan beans is recommended.

Geographical Distribution.- ....."The Plant Disease Survey reports the occurrence of bean blight from New York, Oregon, South Carolina, Tennessee, West Virginia, New Hampshire, Vermont, Massachusetts, Connecticut, New Jersey, Pennsylvania, Delaware, Maryland, Virginia, North Carolina, Georgia, Florida, Alabama, Mississippi, Louisiana, Texas, Arkansas, Ohio, Indiana, Michigan, Wisconsin, Minnesota, North Dakota, South Dakota, Nebraska, Montana, Colorado, New Mexico, Arizona and Idaho. It is also known to be present in Washington and California. It is found in Ontario, and Muncie reports that South American white beans showing characteristic blight discolorations and lesions have been shipped into this country from Brazil. Its presence in Europe is shown by the report of Delacroix, blight being present in bean fields near Paris. It has lately been reported from South Africa, and also from the Philippines.

Economic Importance.- "Blight rivals anthracnose as the most important disease of the bean. The reduction in yield of dry, edible beans, due to bacterial blight was in 1917, estimated at 512,000 bushels, or a yield reduction of 2.48 percent.

"This estimate does not include the blight loss to snap beans, which is also great. It is not uncommon for snap beans examined on the market to show from ten to ninety percent infection."- Rapp, C. W. Bacterial Blight of Beans. Oklahoma Agricultural Experiment Station Bulletin 131. July, 1920. pp. 4-6.

Symptoms of the Disease.

"On the Seed.- In case of light attack, light yellow blotches without definite margins are produced on the seeds, but in cases of severe infection the coloring is deeper and the whole seed may be involved. Infected seed may also be smaller in size and more or less shriveled. The blight infected seed is not to be confused with seed injured by frost. Frosted seed are of a more greenish-yellow or grayish-green color as compared with blighted seed.

"On Seed Leaves.- When such infected seeds are planted and the seedlings appear above the surface of the ground, infected areas may be seen on the cotyledons or seed leaves. Here they are first seen as amber-colored spots or blotches without a well-defined margin. These may enlarge until one or both of the seed leaves are discolored. In some cases the cotyledons are smeared with the yellow, bacterial slime.

"On the Stem.- The bacteria washed down from the cotyledons produce reddish brown cankers on the stem. These lesions are not so deep as those of the bean anthracnose, nor are they so dark in color. These cankers do not have the definite margins, characteristic of the anthracnose spots.

"On the Leaves.- The first appearance of the disease upon the leaves is as small, irregular, water-soaked areas. These areas are more or less puffed up like blisters and may spread until the entire margin or leaf is involved. Later this water-soaked area dries out and turns brown with the collapse of the leaf tissue. The dead areas frequently fall out or the whole leaf is shed. Yellow bacterial masses are frequently scattered over the diseased portion of the leaf.

"On the Pods.- The diseased spots start as small, slightly raised, watery pustules on the pod. These increase in circumference, assuming an amber color. It is not uncommon to see an amber-colored outer area surrounding a slightly raised inner dark green portion. Infection may appear as very small amber blotches, scarcely noticeable on the pod. In the centers of old spots the yellow crusts of bacteria are found. We have noticed that infection is most severe along the hinge of the pod and upon the side exposed to the sunlight. When the bacteria penetrate the pod, yellowed or shriveled seeds may be produced.

"Field Appearance.- When blighted seeds are planted and wet weather is abundant, they may fail to germinate, or seedlings with severely infected seed leaves are produced. This also accounts to a certain extent for skips in the rows.

"In case of severe infection, the field looks as if the plants had been drenched with hot grease, as described in France. The leaves show severe browning or the plants may be completely defoliated. The plants rapidly produce new leaves and blossoming is profuse and irregular. This causes an uneven setting of the pods which cease to fill out or ripen unevenly."- Muncie, J. H. Experiments on the Control of Bean Anthracnose and Bean Blight. Michigan Experiment Station Bulletin 38. December, 1917. Pages 13-14.

Effect of Environmental Factors.

"Weather conditions have an important bearing upon the development and spread of bacterial blight. In our varietal plots the disease always becomes prevalent following a period of warm, wet, muggy weather. Such conditions seem ideal. Until such conditions occur, this disease is of no importance in our plots. Cool, wet weather does not favor its development. Warmth seems essential. Following warm, wet weather, the disease continues to spread so long as the bean plants remain alive, though the spread is not as rapid after the weather becomes dry and the plant tissues harden."- Rapp, C. W. Bacterial Blight of Beans. Oklahoma Agricultural Experiment Station Bulletin 131, p. 17. July, 1920.

Summary

"1. Bacterial blight is a widespread and destructive bean disease, caused by Bacterium phaseoli E. F. Sm.
"2. The bacteria often survive the winter in, or on, the seed, in the soil and on bean straw.
"3. From infected seed the disease is transmitted to the young plant.
"4. Stem girdling may occur.
"5. The disease generally becomes of importance when the pods are developing.
"6. The bacteria generally gain entrance through the stomates.
"7. Cankers upon both leaves and pods are characteristic.
"8. The appearance of bacterial blight as a serious disease is dependent upon weather conditions. Warm, wet weather favors its development.
"9. Spraying has proven of no value as a control measure.
"10. Seed treatments by chemicals, hot water and dry heat are impractical.
"11. Two and 3-year-old seed has, in our work, given blight-free plants.
"12. Foreign seed has not proven of special value. In no case has it been markedly blight-free.
"13. Seed selection is of small value as a control measure. Bacteria are found in seeds apparently disease-free.
"14. Pod selection, though tedious, gives seed free from bacterial blight.
"15. Marked blight resistance has been noted in some varieties. In the development of these varieties along the lines of blight, mosaic and drouth resistance, are the greatest possibilities for permanent control."- Rapp, C. W. Bacterial Blight of Beans. Oklahoma Agricultural Experiment Station Bulletin 131. pp. 37-38. July, 1920.

References.

(1) Duggar, B. M. Fungous diseases of plants, page 119.

(2) Muncie, J. H. Experiments on the control of bean blight. Michigan Experiment Station Bulletin (Technical) No. 38, December, 1917.

(3) Muncie, J. H. A girdling of bean stems caused by Bacterium phaseoli. Phytopathology 7: 306. October, 1917.

(4) Whetzel, H. H. Some diseases of beans. Cornell Experiment Station Bulletin 239, page 210, April, 1906.

(5) Smith, E. F. Bacterial Diseases of Plants. pp. 280-299.

(6) Rapp, C. W. Bacterial blight of beans. Oklahoma Exp. Sta. Bul. 131. 1913.

SOFT ROT OF CARROT AND OTHER VEGETABLES

Caused by bacillus carotovorus Jones

References.

(1) Jones, L, R. A soft-rot of carrot and other vegetables. Vt. Exp. Sta. Report 13: 299. 1901.

(2) Duggar, B. M. Fungous diseases of plants. 1909. p. 131.

(3) Harding, H. A., L. R. Jones and W. J. Morse. The bacterial soft-rots of certain vegetables. N. Y. (Geneva) Exp. Sta. Tech. Bul. 11. 1910.

(4) Stevens, F. L. Diseases of economic plants. (Revised edition). 1921. p. 173.

(5) Smith, E. F. Bacterial diseases of plants. 1920. p. 223.

COMMON SCAB OF POTATO

Caused by Actinomyces scabies (Thaxter) Gussow

Common scab is probably the most widespread and universally prevalent potato disease known.

Laboratory study.

1. Note symptoms and make sketches.
2. If the causal organism is available, study it.
3. Read the following references and write notes.

Discussion.

The following description of symptoms and injury is quoted from Lutman and Cunningham (1).

"Description of the disease.- ..... The malady attacks the tubers during any stage of their development, from the time when they are mere buds until they cease to increase in size, but not, as some authors have believed, while they remain in the ground or after they are placed in storage. The disease first appears as a small brownish spot or stain on the surface of the potato. It was formerly supposed to originate always in the lenticels and there only, but it is shown by one of us (45) to originate on any portion of the potato, although frequently at the lenticels. It is not shown conclusively that deep scabs may arise in this way although the evidence would seem to indicate that they may. The lenticels are composed of cells similar in composition to those of the cork layer, but the intercellular spaces are large and offer an easy avenue of entrance for the fungus. These scab spots, once started, rapidly spread and increase in depth until large patches or the whole exterior of the potato may become affected, leaving very unsightly scars which, in the case of deep or well advanced scab, may penetrate to a depth of a half-centimeter. These spots consist of accumulated corky tissue which may be readily removed, leaving the unprotected inner parenchyma tissue exposed in a manner closely resembling the condition which exists when a scab has been removed from injured animal tissue. The diseased cells lose their starch and masses of globular bodies appear, which, according to various authors, may be disorganised tissue or fat globules. The fact that the mass of scab tissue may be eaten by wire worms or insects, and that these animals are often found in the affected spots, has given rise to the popular but erroneous impression that scabby potatoes are caused by their "bites". The scab is the result of the attempt of the cork cambium to protect the inner starch-bearing parenchyma tissue against the irritating action of the invading organism by the production of the new cork cells beneath the diseased part. The cork cells thus formed are abnormal in form and in the thickness of their walls. Some of them are strongly hypertrophied and expanded while others are crushed and distorted. It would seem as though there were a series of formations of this nature in view of the fact that the abnormal growths show more or less distinct layers. Frank and Krüger (21) claim that in certain types of scab a hyperplasie of the parenchyma underneath the cork occurs and that this bulge in the starch layer causes the protuberance of the spot. They claim to be able to distinguish four different types of scab, as follows:

(1) "Shallow scab, the diseased portion being on a level superficially with the remainder of the corky layer of the tuber.

(2) "Deep scab, the diseased tissue sinking into the body of the tuber, a hole filled with the abnormal cork being formed.

(3) "Bulging scab, wherein the abnormally stimulated parenchyma pushes the cork tissue out beyond the level of the normal skin of the tuber.

(4) "Bulging deep scab, a combination of the deep and bulging forms, wherein the parenchyma is affected at the infected areas and grows abnormally, but at the same time the cork cambium sinks into this parenchyma producing a spot which is deep but also bulges beyond the level of the surrounding cork.

"The types established by Frank and Krüger are not distinct and all sorts of intermediate gradations could be picked out of a bin of scabby potatoes. Freshly dug potatoes often show the scabbed portions slightly raised above the surrounding tissue. This is due to an increase in the amount of parenchyma beneath, but after the tubers have thoroughly dried for some time in the bin this appearance is not as noticeable.....

"Injury caused by scabbing.- The vital practical question in any plant disease problem is the actual loss to producer and consumer. The potato is an almost universal article of food. Fluctuations in the volume of the crop necessarily effect the cost to the consumer. It seems scarcely possible that potato growers and consumers recognize the vast extent of the loss caused by this malady. It is the popular notion that the injury and loss falls only on the grower and is confined to the depreciation in value due to an unsightly appearance which lowers sales prices. But the consumer also suffers. He may buy at a lower gross price, but on the other hand he experiences a considerable loss of food material otherwise available, due to the deep paring needed to remove the scabs when preparing the potato for the table. Moreover, some buyers object seriously to the peculiar earthly odor and taste which is characteristic of scabbed but is foreign to unscabbed potatoes. This quality, however, is not disagreeable to all people; indeed, many prefer scabbed potatoes because of this very odor and taste and because of their frequently increased mealy texture. This last quality is decidedly enhanced by severe scabbing. Neither taste nor mealiness are objectionable to the writers; and they have known many people who were exceptionally fond of scabby potatoes.

"Beckwith (2) concluded that the use of scabby potatoes as seed decreases neither the number nor the yield of tubers but, rather, that if any effect whatever was secured the total production was greater as a result, although naturally the percentage of scabbed specimens was increased. His observations are not in line with those made elsewhere. Thus Bolley (5) in 1891 states that 'the farmer is unable to judge how much greater the yield in bushels per acre might have been had the crop not been affected'. The next year he cited the results of experimental trials tending to discredit the theory that increased yields followed the use of scabby seed. He stated that 'while it is a common assertion on the part of potato planters that the disease does not affect the yield, that it is more often associated with a heavy yield, I am enabled from the consideration of numerous experiments to say that the idea is wholly erroneous. In fact the yield of any given hill is always diminished..... A disease which lessens the yield from fifty to sixty bushels per acre, aside from the fact that the entire product is made unsightly and open to excessive waste in preparation for the table, is worthy of no slight effort at prevention'."- Lutman, B. F. and G. C. Cunningham. Potato Scab. Vermont Agricultural Experiment Station.Bul. 184 6-11. September, 1914.

Influence of soil temperature on scab.

In Wisconsin Research Bulletin 73 is given an account of some experimental work conducted to determine the influence of soil temperature upon the development of potato scab. The summary from that bulletin follows.

"Summary.

1. "The development of potato scab, caused by Actinomyces scabies, is evidently influenced by several environmental factors. An attempt has been made to secure evidence as to such possible influence of soil temperature.....

2. "The disease developed at all soil temperatures, 11° to 30.5° C., but was comparatively slight at either extreme.

3. "The optimum soil temperature for scab development as measured by the number of scabby tubers was found to be about 23°C., while the optimum for the percentage of the total tuber surface scabbed was a little lower, about 20.5°C. The conclusion reached is that all things considered 22°C. may be accepted as about the optimum soil temperature for scab development, where the 'Wisconsin tank' method is used.

4. "A field trial was also conducted in which three gradations of soil temperature were maintained during the season of tuber development. Of these the highest, approximately 25°C., proved to be the most conducive to scab development.

5. "Field observations seem in general to accord with the results obtained by experiments. They indicate that potato scab is comparatively more prevalent in regions having high summer temperatures than in those of lower temperatures, and also that in the same district in Wisconsin the disease development is greater during hot than during cool summers. It is to be noted, however, that such observational data is relatively meager and is to be considered only as suggestive. Attention should be directed to this question by other observers.....

6. "The available evidence indicates that the scab parasite as an independent organism is favored by relatively high temperatures, whereas the potato plant functions better at relatively low temperatures. It is, however, noteworthy that the influence of temperature upon the different potato organs is not uniform and that it varies also with the stage in their development.

7. "The influence of temperature upon the prevalence of the parasite in the soil may be cumulative from season to season whereas the influence upon the host is immediate and temporary.

8. "The immediate relation of temperature to the development of scab seems to be more closely correlated with its fluence upon potato tuber development than with that upon the growth of the parasite.

9. "It seems evident that a satisfactory interpretation of the relation of soil temperature to the development of potato scab must await on the one hand more critical study of its relation to tuber development and on the other hand a fuller knowledge of the details as to tuber infection and subsequent scab development."- Jones, L. R., H. H. McKinney and H. Fellows. The Influence of Soil Temperature on Potato Scab. Agricultural Experiment Station of the University of Wisconsin Bul. 55: 33-34. July, 1922.

Control Measures

Brann and Vaughan (6) recommend the following control measures.

"Corrosive Sublimate.- 4 ounces to 30 gallons of water, has proved the best disinfectant for controlling potato scab..... It is also the best control for Rhizoctonia and black leg. Since the chemical dissolves slowly in cold water, it should first be stirred into about a gallon of hot water.

"The strength of the corrosive sublimate solution can best be maintained by adding $\frac{1}{2}$ ounce for every 4 bushels of seed potatoes treated and keeping the volume of water constant. This additional amount is based on the $1\frac{1}{2}$ to 2 hour treatment. If a shorter treatment is given, a correspondingly smaller amount of chemical should be added. For example, if 4 bushels are soaked 30 minutes, add 1/8 ounce of corrosive sublimate. For convenience have the corrosive sublimate in a stock solution, strength 1 ounce to 1 quart of water, made up in fruit jars or stone crocks.

"Treat only sound, uncut tubers before the sprouts have started. Dry the tubers at once after treatment to prevent continued action of the chemical. In case of damp, slow-drying weather the tubers should be rinsed with clean water immediately after treatment. Keep in clean crates or sacks in half light for 10 days to two weeks before planting. This prevents reinfection and allows strong sprouts to start. Do not place the potatoes in direct sunlight because they will get too hot and develop black heart.

"Barrels, or wood or cement tanks are frequently used in treating potatoes. The tubers may be handled loose or in crates or sacks. If sacks are used they should first be washed and disinfected; this prevents weakening of the disinfectant. Galvanized tanks may also be used if the interior is coated with asphaltum paint.

"Cautions.- Corrosive sublimate is a deadly poison if taken internally; therefore, be very careful to keep it away from young children and all farm stock. Avoid over treatment either by too strong a solution or too long a time, as the eyes of the potato may be injured and germination retarded. Do not use treated potatoes for food.

"Select Scab-Free Tuber for Seed.- The greater the amount of scab on the seed, the greater will be the number of scab germs introduced into the soil, and the greater the amount of scab on the crop. The use of "leftover" and warehouse seconds is strongly discouraged.

"Practice Crop Rotation.- The best growers have demonstrated that it is not advisable to grow potatoes on any given field oftener than every third year. A rotation with grain and clover is desirable where a part of the cultivated crop may be corn. The plowing under of a crop of clover or fall rye before planting potatoes is a practice to be highly commended from the standpoint of maintaining the fertility and good physical condition of the soil and in the control of scab.

"Select Scab-Free Soil.- Soils in different sections of the state, on different farms, and even in different fields of the same farm vary in the amount of scab organisms present. In general, the older sections and fields long used for potatoes will have the most scab. A farm plan will greatly assist in locating fields where potatoes should or should not be planted.

"Avoid Heavy Applications of Lime, Wood Ashes, or Manures Just Previous to Planting.- If the soil is too acid and needs lime, it should be applied after rather than before the potato crop. Good results have been secured by applying manures to the clover field the year previous to using it for potatoes.

"Avoid Feeding Uncooked Scabbed Potatoes to Stock.- Experiments have shown that the scab organism may pass through the digestive tracts of animals without losing its power to produce the disease."- Brann, J. W. and R. E. Vaughan. Potato Scab. Agricultural Experiment Station of the University of Wisconsin. Bul. 331: 6-9. April, 1921.

Review

Compare common scab and powdery scab in all details of symptoms, life history and control.

References.

(1) Lutman, B. F. & Cunningham, G. C. Potato Scab. Vermont Exp. Sta. Bul. 184. 1914.

(2) Maney, T. J. Effect of potato scab treatment on seed vitality. Iowa Exp. Sta. Bul. 148. 1914.

(3) Lint, H. C. Seed and Soil treatment for Control of potato scab. New Jersey Exp. Sta. Cir. 95. 1918.

(4) McKay, M. B. Control of Potato diseases in Oregon. Ore. Agr. College Extension Bul. 186. 1917.

(5) Lutman, B. F. Resistance of potato tubers to scab. Vermont Exp. Sta. Bul. 215. June, 1919.

(6) Brann, J. W. and R. E. Vaughan. Potato scab. Wisconsin Exp. Sta. Bul. 331. April, 1921.

(7) Jones, L. R., H. H. McKinney and H. Fellows. The influence of soil temperature on potato scab. Wisconsin Exp. Sta. Research Bul. 53. July, 1922.

CHAPTER XVIII

Diseases caused by fungi - Phycomycetes.

By far the greater number of parasitic diseases of plants are caused by fungi. Fungi are thallophytes which have no chlorophyll and consequently cannot carry on the work of photosynthesis. Therefore all fungi are dependent plants and live either saprophytically or parasitically.

One of the most striking differences between fungi and the two groups of parasites previously studied (Slime Moulds and Bacteria) is found in the vegetative phase, which, in fungi, consists of branched fungous threads, the hyphae, which, considered collectively, make up the mycelium.

Reproduction in fungi normally takes place by means of spores produced either sexually or asexually or both.

In this outline the fungi will be considered as divided into four classes, namely, Phycomycetes, Ascomycetes, Basidiomycetes and Fungi Imperfecti.

DISEASES CAUSED BY PHYCOMYCETES

The Phycomycetes are characterized by their alga-like reproductive habits and their non-septate mycelium. Many serious plant diseases are caused by fungi belonging to the phycomycetes. Probably those diseases caused by the "Downy Mildews", one of the most important families of the Phycomycetes, are the most numerous and harmful of the diseases caused by this class of fungi. The general term "blight" is quite frequently applied to diseases caused by the downy mildews as well as to diseases caused by other groups of parasites.

CROWN WART OF ALFALFA

Caused by Urophlyctis alfalfae. (Lagerh.) P. Magnus.

This disease was first reported from South America in 1892. It was later discovered in Europe (1902) and in 1909 was first reported in the United States from California. It is now known to occur in several regions of the United States including Oregon.

In severe cases brown, dead stems and weak, yellow-leaved stems may be found in the field but the most typical symptom is the production of gall or wart-like outgrowths at the base of the stems.

1. Laboratory.

a. Symptoms. - Examine specimens and make sketches of the diseased parts. Describe symptoms in notes.

b. Pathological histology.- Examine sections of a wart and draw to show effect of disease on tissues of host. What effect has the disease on the cell structure.

c. Life history of causal organism.- Look for spores of the fungus imbedded in the tissues of the host. Represent the fungus in the drawing made for (b) above.

d. Write a description of what you have observed, then consult the references given below and complete your notes for this exercise.

Text discussion.

The following account of this disease is taken from an article by Jones and Drechsler (5). Parts of the discussion are quoted and other parts are abstracted briefly.

Common Names.- "In the United States the disease is commonly known by either of two names, crowngall and crownwart. As will be shown later, the structure of the diseased tissue is that of a true gall, and it was called such in the earlier reports of its occurrence. Later the name crownwart was suggested in order to distinguish the disease from the bacterial crowngall caused by Pseudomonas tumefaciens, though it had not then been shown that this disease occurs upon alfalfa in the field. Recently, however, galls have been found by Mr. H. L. Westover on alfalfa in Arizona which appear to be true crowngalls, though complete proof is lacking. In view of the fact that a gall similar in appearance to that caused by Urophlyctis alfalfae (Lagerh.) P. Magnus is found upon alfalfa, it is even more desirable than formerly that the disease caused by Urophlyctis should have a distinctive name. The fact that the name crownwart is well established in usage is much in its favor. It will be seen, however, from facts presented later in this paper that this name is somewhat misleading, inasmuch as the galls are not typical warty growths, nor are they formed from the tissue of the socalled crown of the plant in a manner comparable with that in which crowngalls are formed....."

Distribution.- The disease has been reported from Ecuador in South America and from Germany, Italy, France, Holland, Sweden and England in Europe. In the United States it has been found abundant only west of the Sierra Nevada and Cascade Mountains, though it has been seen east of these mountains. It has been reported from Oregon, Utah, Arizona and California.

Economic importance.- There seems not to be any definite information as to the amount of damage resulting from crown-wart. Some observers report considerable damage while others state that some fields show much disease without a corresponding reduction in yield. Jones and Drechsler do not attempt to estimate the losses resulting from this disease.

Description of the Disease.- "The disease is more easily described by stating briefly the origin and method of development of the galls. So far as the writers can discover all galls as they occur naturally in the field result from the infection of buds in early stages of development as they emerge from the crown of the plant. It is well known that there is an almost continuous succession in the development of buds from the so-called alfalfa crown during the entire year. A portion of those buds which will produce the shoots furnishing the first crop in the spring have begun development as early as the preceding autumn. Generally speaking, the first buds to be formed in the seasonal succession have a point of origin deeper in the soil than those which are formed later, so that many of the buds from which the shoots of the third cutting arise develop from positions quite above the surface of the soil. Buds produced below the soil level in cool weather appear to have a meager protection of scaly covering, and it is for the most part such buds that become infected and give rise to galls. Thus, galls are swollen and distorted bud elements, scales, leaves, and stipules.

"Unless overwintered galls which are described later are discoverable, the disease is first evidenced in the spring by a slight thickening and rounding of the young buds. During two years this has been observed near Chico, California in the latter part of March or early in April. The diseased buds become more and more rounded as growth progresses and are glistening white in color. Then, as the infected structures begin to push apart, some of them grow much more rapidly than others until the structure as a whole assumes a conspicuously irregular form. In most cases, however, an examination of the gall will show that it is made up of thick, scale-like layers about a central growing axis. Sometimes this axis continues growth in spite of the demands of the mass of developing gall tissue and produces a weak shoot. The earlier and more vigorous buds produce the larger galls. Smaller galls often appear to be developed from smaller buds along the stems below ground that would ordinarily remain dormant. The origin of galls that appear on stems several inches above the surface of the ground in wet weather appears to be due in part to the infection of axillary buds that would never develop in the ordinary course of events and in part to the elongation of the stems and petioles which force infected tissue upward.

"Since a large part of the infected buds are developed at a depth of 2 or 3 inches below the surface of the soil, the majority of the galls are so far below ground that they escape observation unless the soil is removed from around the plant. If they are of large size some of them come to the surface, where they take on a green color and in extreme cases form a crust of diseased tissue around the base of the healthy stems.

"Another type of gall that is not common results from local infections on young leaves. Such infections give rise to small blister-like galls much like those produced on Sanicula spp. by another species of this fungus which will be mentioned later.

"The galls reach full development early in the summer, in early June in northern California. From this time on the majority of them begin to decay if moisture is abundant, or to shrivel and dry with the coming of drouth. However, in almost all fields a few galls more deeply situated become covered with a corky layer and survive the winter.

"When plants are subjected to dry conditions in late summer, as they usually are in the Rogue River valley in Oregon, many of the galls do not decay but remain living throughout the autumn and winter. It does not appear that such galls make appreciable growth during the following year. Nevertheless, gall tissue may accumulate around old plants in considerable mass. The exterior becomes covered with a brown, corky layer that has a much warted appearance. This accumulation of gall tissue has not been found on plants that have grown in well-irrigated fields.

"At whatever age or state of development these galls are found, they possess one distinctive character that is discovered when they are cut open. The interior of the galls contains many small, irregularly shaped brown masses of fungus spores which are easily visible. In old dried galls the host tissue has shrunken so much that the spores mass often occupies a large portion of the mass of the gall. Even in decayed galls that have not yet been broken to fragments the spore masses can be recognized by their golden brown color.....

Morphology of the fungus.- "The thallus of the fungus consists of two types of structures, turbinate cells and resting spores. In the first turbinate cell that is the immediate development of the infecting fungus are inserted a number of septa which delimit uninucleated peripheral segments from a polynucleated central sporogenous mass. A hyphal structure of limited growth develops from each of these segments and carries the nucleus in its expanded termination, the latter constituting a young turbinate cell of the next succession. At its mature stage the turbinate cell bears a branched apical haustorium, the short axial element of which proliferates at its tip a globose terminal expansion into which the polynucleate sporogenous mass of protoplasm migrates to produce the resting spore. This is characterized by 9 to 15 branched haustoria in zonate arrangement between the equator and the distal pole.

"A solution of the thinner cell walls in proximity to the young, advancing turbinate cells leads to the development of cavities in the hypertrophied tissue in which the resting spores are finally found inclosed.....

Germination of the Resting Spores.- "As has long been recognized, the fungus passes through the prolonged periods of summer drouth by means of the resting spores contained within cavities in the galls of the host. In the course of the rainy season the galls disintegrate completely, thus setting free the spores; and it is not improbable that the exposure incident to this method of liberation may be necessary for germination. However, the conditions that may favor germination remain more or less obscure.....

"Infection appears to take place only early in the spring, becoming easily discoverable in the latter part of March or in early April in northern California.

"In irrigated regions, or in regions where there is abundant moisture during the entire season, most of the galls reach full development early in the summer and thereafter decay rapidly, only a few surviving until the next spring.....

"The abundant development of the disease in the regions where it now occurs is apparently associated with excessive moisture during the period when infection is taking place. Any measures which can be taken to reduce the moisture near the

surface of the soil at this time should reduce the disease." - Jones, Fred Ruel, and Charles Drechsler. Crownwart of Alfalfa Caused by Urophlyctis Alfalfae. Journal of Agricultural Research 20: 320-321. November 15, 1920.

References

(1) Smith, Elizabeth H. A note on Urophlyctis Alfalfae in California. Science, N. S., Vol. 30, No. 763. August 13, 1909.

(2) McCallum, W. B. (Plant Physiology & Pathology). Arizona Exp. Sta. Report, June 30, 1909.

(3) O'Gara, P. J. Urophlyctis alfalfae, a fungus disease of alfalfa occurring in Oregon. Science, N. S. 36: 487. 1912.

(4) Jackson, H. S. Alfalfa and clover diseases. Oregon Exp. Sta. Biennial Crop Pest and Horticultural Report, 1911-12, p. 300.

(5) Jones, F. R. and Charles Drechsler - Crown wart of alfalfa caused by Urophlyctis alfalfae. Jour. Agr. Res. 20: 295. November 15, 1920.

POTATO WART

Caused by Chrysophlyctis endobiotica (Schibb.) Perc.

This serious potato disease which has been known in Europe since 1896 made its appearance on this side of the Atlantic in 1909 when it was discovered in Newfoundland. In 1918 the disease was discovered in certain sections of Pennsylvania. It is feared that unless proper precautions are taken the disease will spread until it becomes a menace to the potato industry of this country.

1. Examine specimens of diseased plants and also illustrations and make sketches to show symptoms. Describe in notes.

2. If sections of diseased tissue are available make a study of the relation of the fungus to the host and illustrate by means of drawings.

3. Read the following references and write complete notes on geographical distribution of the disease, symptoms, life history of causal organism and control measures.

Text discussion.

The following brief description is quoted from Orton and Kern (1).

"A most serious disease of the potato, which is well described by the common name, Potato Wart, has been found to be fairly well established in Luzerne County,

Pennsylvania. This disease which has been also called Black Wart, Black Scab, Warty Disease, Cauliflower Disease and Potato Canker, has been known to science since 1896, when it was described in Hungary. The first published reports of its presence in Great Britain appeared in 1902, but there is evidence to show that it was present many years without being recognized. It was certainly there in 1899 and possibly twenty years before that. Since that time it has become widely distributed in districts in England, Wales, Scotland and Ireland. In 1909 the disease made its appearance on this side of the Atlantic Ocean, having been discovered in Newfoundland by the Dominion botanist.....

"The infested district in Pennsylvania is a mining region, there being no land under cultivation except the gardens, the produce of which is grown for home consumption, and there are no contiguous cultivated areas. It does not seem likely that potatoes, garden truck, or other plants or plant products, which might carry the disease, will be sent out of the region, and yet the disease has been so virulent and has spread so rapidly in other countries where it has become established, that it seems advisable to spread widely the news of the situation, so that all potato growers may know of the possible danger.....

"Symptoms of the Disease.- In its usual development the disease is characterized by prominent wart-like outgrowth on the tubers. These warty excresences on the tubers are responsible for the common names "potato wart", "warty disease", "canker", and "cauliflower disease". The warty growths vary in size and distribution. For the most part they appear on the tubers, having their origin in the eyes, but they may occur on the sprouts which develop new tubers, on the lower portions of the stem, and possibly on the roots. The warty masses are often suggestive of the crown gall disease known on various plants. Being entirely underground the disease is not evident until digging time, although some observers have pointed out a tendency for the tops of badly diseased plants to remain green longer than in the case of healthy or only slightly affected plants.

"The warts are at first whitish or of the color of the young tubers, but eventually they turn to a darker shade, a fact which is responsible for the name "black wart" in some localities. When disturbed and left exposed on the ground, the warty masses may continue growth and often take on a green color. The warty tumors vary from the size of a pea to growths which may equal or exceed the size of the tuber from which they arise. Where smaller growths occur the remainder of the tuber may appear quite normal. In severe cases the whole surface of the tuber may become involved, thus presenting a large warty mass which has lost all resemblance to a potato. In diseased hills some tubers may appear on cursory examination to be healthy, but the eyes are likely to be infected. In the early stages, it may be difficult or even impossible to recognize the presence of the disease, and it is by the use of such apparently sound tubers for seed that the disease has been spread in the regions where it has been introduced."

Morphology and life history.- "..... It is certain that this fungus does not develop at any time a mycelium of thread-like elements, such as form the vegetative phase of most fungi. The fungus lives entirely within the host cells. An examination under the microscope of a bit of the diseased tissue taken in the autumn from one of the warts, reveals the fungus in the host cells just below the surface. In infected cells may be seen conspicuous rusty brown bodies which are the reproductive organs of the parasite. The brownish structures are globoid or

oval in shape and when mature have a thick outer wall for the protection of the contents. The wall is rough with somewhat irregular ridges.

"By the decomposition of the rather fragile host tissue comprising the warts, these bodies escape into the soil and it is in this way that the soil becomes infested. These heavy walled bodies are a resting condition of the fungus tiding it over from one growing season to another. It is certain that they may remain alive for several years. It is reported on good authority that soil containing them may retain its infecting power for six to eight years. For a long time the form of activity which these resting bodies assumed when they produced new infections was obscure. All investigators now agree that they divide internally into numberous spores which finally possess the power of moving about, hence known as swarm spores. There is a great deal of uncertainty as to the manner in which these gain entrance to the potato cells. We have had no opportunity to make observations on this point. It is certain that these swarm-spores are the simplest stage in the life history of the fungus, and that they are the actual infecting agents. This makes it evident that the heavy-walled bodies are spore cases, or sporangia.....

"After entry into the host the swarm-spores of the parasite become imbedded in the living cytoplasm of the host cells. Several uninucleated bodies which are apparently swarm-spores may be observed in a single host cell, frequently as many as twelve or more having been observed. They show a very marked tendency to cluster around the nucleus of the host cell. Whether these parasitic bodies may migrate from one host cell to another is not clear....."

"Preventive Measures.- For the prevention of any disease there are four lines of attack, namely, exclusion, eradication, protection and immunization. The attempt to exclude potato "wart" from the United States has failed, because the disease was, in all probability, introduced into our midst before the Federal quarantine was placed against it. It is possible, however, to exclude the disease from other localities in the state and from other states by placing a guard in the form of a strict quarantine around the infested region. This will be done promptly by the Bureau of Economic Zoology of the State Department of Agriculture, which office by law controls the inspection and movement of nursery stocks and other plant materials. Any regulatory actions must receive the heartiest support from all concerned if this terrible disease is to be held in check.

"No method of eradicating the disease has been attempted on a large enough scale to enable us to judge whether or not this can be done. As the parasite is known to live in the soil for several years, at least under some conditions, the process of starving it out is manifestly a slow one. Whether soil sterilization with steam or chemicals may be efficient remains to be proved.

"There is no way known of protecting potato plants from infection by this fungus by spraying, seed treatment, and the like. Therefore, potatoes must not be grown in infested soil until such soil has been shown to be free of the parasite.

"In the British Isles considerable advance has been made along the line of obtaining varieties of potatoes immune to potato "wart". Several varieties have been found which show a remarkable degree of resistance to this disease. It is our intention to secure the most promising of these varieties and test them out

under our conditions along with the leading varieties already grown in this country. It is to be hoped that some variety will be found which is immune to this disease.

"As the fungus no doubt survives the digestive and alimentary tracts of animals and may be spread in the manure, it is extremely important that any diseased tubers or peelings from such tubers, be boiled before feeding in order to kill the fungus. It is also quite apparent that the habit of throwing potato peelings on the garden for the poultry to pick up has been one of the commonest methods of spreading this disease in this state and is one which should be discontinued forthwith. If it is desired to feed such refuse it can be made safe by boiling. If not used for feeding purposes it should be completely destroyed by burning.

"In view of the national and international importance of potato "wart" it is the duty of every person who grows potatoes, whether in the field or garden, to be extremely cautious to use for seed only tubers which are healthy in every detail of appearance. Slight infections are easily overlooked and great care must be taken. Anyone observing suspicious cases should feel it their duty to report them at once, so that expert advice can be given. Only by such cooperative spirit can we hope successfully to combat and eradicate potato wart." - Orton, C. R. and F. D. Kern. The Potato Wart Disease. Pennsylvania Agricultural Experiment Station Bulletin 156. March, 1919.

References

(1) Orton, C. R. and Kern, F. D. The potato wart disease. Penn. Exp. Sta. Bul. 156. March, 1919.

(2) Gussow, H. T. A serious potato disease occurring in Newfoundland, Bul. 63, Dep't. of Agriculture Central Experimental Farm, Ottawa, Canada. Oct., 1909.

(3) Orton, W. A. Wart disease of the potato. U.S.D.A. B.P.I. Cir. 52. March 7, 1910.

(4) Kunkel, L. O. Black Wart. Plant disease Bul. issued by the Plant Disease Survey U.S.D.A. B.P.I. 2: 197. October 1, 1918.

(5) Sanders, J. G. The discovery of European potato wart disease in Pennsylvania. Jour. Econ. Entom. 12: 86. 1919.

DAMPING-OFF.

Caused by Pythium debaryanum Hesse.

There are several different species of fungi which cause damping-off of plants, especially seedlings. Some of these belong to other classes of fungi and will be mentioned in following Chapters. Species of Corticium (Rhizoctonia), Fusarium, Sclerotinia and Botrytis are known to cause damping-off at times. (See chapter X)

However the subject of this exercise, Pythium, is one of the oldest and best known fungi causing this type of trouble.

Laboratory Study.

a. Symptoms:- Examine specimens of seedlings, tomato, egg-plant, conifer seedlings, or any other available material showing this type of disease. Note the point of attack, nature of injury and extent of harm done. Make sketches to illustrate symptoms, and describe in notes.

b. Morphology and life history.

(1) Asexual stage of reproduction.- This consists of Conidia. If material is available examine under microscope and draw.

(2) Sexual stage.- This consists of oogonia and antheridia. Fertilization results in an oospore. Mount a specimen under the microscope and draw.

c. Notes.- After reading the following discussion of the disease write up complete notes.

Text discussion.

"This disease is very familiar to every grower of plants. The trouble is peculiar to seedlings or very tender plants. It is prevalent in the greenhouses, the hotbed, the cold frame, and frequently also in the field. The trouble is induced by the presence of definite parasitic fungi in the soil. They thrive best when the land is continually damp and the atmospheric temperature comparatively high. Damping off is also favored by thick sowing and too much shade in the seed bed.

"Symptoms of Damping off.- Every experienced trucker knows the disease when he sees it. Seedlings freshly damped off are soft and water-soaked at the base of the stem. If they are pulled they often break off easily. A more careful examination shows that the root system is entirely decayed by this time, although the upper part of the stem and leaves may still be green, possibly also fresh. The degree of prostration in the seedlings is regulated by the amount of moisture in the soil. If the amount of moisture is slight, the seedlings will be flabby and wilted before they topple over. With a high moisture content, they are more firm, but become prostrate as soon as infection starts in. Damping off usually begins in spots in the seed bed or in the field and then may spread in every direction.

"The organism.- Pythium de Baryanum was first named and described by Hesse in 1874. Ward found it to be a very prevalent parasite in the garden soil of Europe. In America the fungus was first recognized by Atkinson as of great economic importance. Pythium de Baryanum, when examined under a compound microscope, is seen to be made up of coarse, non-septate, highly granular, irregular branched hyaline vegetative threads or mycelium. The younger threads are more finely granular, the

oldest ones are coarsely granular or more often empty. These threads penetrate the cells of the host, where they obtain food.

"Pythium de Baryanum does not often fruit freely on the dead host. The fruiting is better observed when it is grown in a pure culture. Under normal conditions the fungus produces two forms of spores, conidia and oogonia. The summer spores, or conidia, are swellings formed at the tip of the hyphae. These swellings readily break off from the mother threads and germinate by sending out a slender tube. This tube penetrates the seedlin tissue, where it grows and develops and after due incubation reproduces the disease. The oospore or sexual spore is the stage which is most commonly found. The female oogonium first develops as a terminal enlargement which is cut off by a septum from the mother thread. Next or adjacent to it a slender tube is cut off from the mycelium by a septum. This tube now performs the function of the male sexual organ and is known as antheridium. The latter then comes into close contact and empties all its content into the oogonium. Fertilization thus takes place, and a mature egg or oospore or winter resting spore is formed.

"The latest investigations have not yet disclosed whether or not Pythium de Baryanum is carried over from year to year by its oospores. It is apparently able to propagate itself indefinitely by its vegetative mycelium."..... - Taubenhaus, J. J. Disease of Truck crops and their Control. 1918. pp. 42-44.

Control.

There are three methods that may be used in controlling damping-off, namely, (a) soil disinfection, (b) crop rotation and (c) manipulation of the environment.

The soil may be sterilized by the use of formaldehyde or other chemical such as sulfuric acid, or by the use of steam or hot water, (See Chapter VII.).

Any disease caused by an organism which lives in the soil can be combated by keeping the susceptible crop off the infected soil until the parasite has been starved out.

Usually damping-off fungi are very sensitive to temperature and moisture relations and where these can be controlled, as in the greenhouse, the disease may be lessened or eliminated by avoiding excessive humidity and high temperature. (See Chapter X).

References.

(1) Taubenhaus, J. J. Diseases of Truck Crops. 1918. pp. 42-44.

(2) Stevens, F. L. Diseases of Economic Plants (Revised Edition) 1921. pp. 19-20.

(3) Duggar, B. M. Fungous Disease of Plants. 1909. pp. 141-144.

(4) Johnson, J. The control of damping-off disease in plant beds. Wis. Exp. Sta. Research Bul. 31. 1914.

(5) Hartley, Carl. Damping-off in forest nurseries. U. S. D. A. Bul. 934. 1921.

(6) Gifford, C. M. The damping-off of coniferous seedlings. Vermont Exp. Sta. Bul. 157. 1911.

THE DOWNY MILDEW FAMILY (PERONOSPORACEAE)

This family of fungi ranks high in economic importance. Members of this family usually exhibit the reproductive features characteristic of the Phycomycetes in general. The sexual stage consists of oospores imbedded in the host tissue. The asexual stage consists of variously branched conidiophores and conidia borne aerially on the surface of the host. The "Downy Mildews" should be compared critically with the "Powdery Mildews", a family of the Ascomycetes, when that family is taken up later in this course.

Four "Downy Mildew" diseases have been selected for study in the following pages, namely, Downy Mildew or Late Blight of potato, Downy Mildew of lettuce, grape Downy Mildew and Onion Downy Mildew or Blight.

LATE BLIGHT OF POTATO

Caused by Phytophthora infestans. (Mont.) De Bary.

This is a very serious potato disease in some parts of the country whenever the conditions are favorable for its development. (Find out what these conditions are). The fungus causing this disease belongs to the group known as downy mildews.

Laboratory.

a. Symptoms.- Late blight attacks both the foliage and the tubers of the potato plant.

(1) Foliage.- Examine diseased leaves both dried and preserved in liquid and make sketch to show symptoms. When leaves showing the fruiting stage are freshly collected they have a downy appearance.

(2) Tubers.- Examine diseased tubers which have been preserved in liquid preservative and sketch. Look at illustrations and read descriptions of symptoms on all parts of plant in Duggar's text and in the bulletins listed below. Write up complete description of all symptoms.

b. Morphology and life history of causal organism.- The vegetative phase of this fungus, the mycelium, grows within the host tissues and only the fruiting stage, the conidiophores and conidia appear on the surface of the leaves. Examine

with microscope small bits of the leaf mounted on a slide and find the conidiophores protruding from the leaf surface. They emerge through the stomata. Can you observe this fact? Note the manner of branching and any other distinguishing characters of the conidiophores. Find conidia attached to branches of conidiophores. If none are attached, find detached conidia. Conidia germinate by production of zoospores. Make a drawing to illustrate the fruiting structure.

c. Notes.- Write such notes as you can from the material examined in the laboratory, then consult the following references and finish your notes. You should write a complete report on this disease following the general outline for such report.

Text discussion.

The following account of late-blight is quoted from various sources, namely, Duggar (1), Jones, Giddings and Lutman (4) and Melhus (7).

"Symptoms.- Upon the leaves of the potato this fungus develops characteristic spots which cannot be easily confused with other potato diseases. These spots frequently begin at the edge or tip and spread until the whole leaf may be involved. They present in moist weather a dark, somewhat water-soaked appearance with slightly purplish tint. In drier weather they are brown without the definite markings of the early blight. The moist appearance of the spots accompanied by the wilting of the leaf, or of that portion affected, offers an easy diagnosis. Generally there is no accompanying stem injury, but in some cases the trouble may extend to the stem; or, again, it may be found upon the leaves as an extension of a stem affection. Upon the tubers this fungus develops the well-known dry rot. On account of the presence of the mycelium within the tissues of the tuber the cells are killed and the tubers rendered liable to the ordinary forms of wet rot induced by bacterial action or by mold fungi. The dry rot may cause serious damage in the field, yet this damage may be further emphasized or even first made evident while the potatoes are in storage."- Duggar, Benjamin Minge. Fungous Diseases of Plants. Page 167.

"The fungus.- The mycelium of the Phytophthora, like that of the other members of this family, is unicellular, and the haustoria are filamentous. The conidiophores arise singly or in groups of from two to four from the stomates. The conidiophore is branched, and at the tip of each branch a conidium is produced. The conidium is pushed to one side and the branch continues. The continuation is, however, larger than the tip which produced the conidium, so that this further growth is marked by an enlargement of the branch, making a very characteristic form of conidiophore. The conidia are ovate and .. germinate readily when fresh, by the production of about eight zoospores. Germination may be secured in water but apparently not in nutrient solutions. The zoospores are motile for a brief time, perhaps seldom longer than an hour. They then come to rest and appear spherical and invested with a wall. Germination readily follows, and the germ tube penetrates the leaf either by stomates or by boring through the cuticle. The conidia serve not only to spread the disease rapidly from leaf to leaf, but they also fall upon the soil and may be brought in contact with tubers. They

enetrate the tubers as readily as the leaves, the dry rot being produced in con-
equence."- Duggar, Benjamin Minge. Fungous Diseases of Plants. Pages 169-170.

"Bodies having the characters of thick-walled spiny resting spores were pro-
uced in pure cultures in potato gelatin and in Lima-bean agar. In the early
tages of their development these bodies had the general appearance and the cyto-
ogical characters of oogonia, but no antheridia were found, and they apparently
eveloped asexually. Nine strains of the fungus from widely different sources
n America and Europe have been studied with reference to the production of these
odies. They were found in all. Growth in mixed culture did not increase or
odify them. Persistent search failed to reveal the mature stages of these bodies
n cultures upon blocks cut from potato tubers, although what were considered to
e immature stages of the same bodies were found sparingly. No such bodies have
een found in decaying potato tubers following Phytophthora attacks. Examination
f potato leaves killed by Phytophthora showed that somewhat similar bodies occur.
ome of them clearly originated from secondary saprophytes and probably all so
riginated. No opinion is therefore justified as to the occurrence of such resting
pores in nature, although their appearance in the culture tubes proves that
hytophthora is capable of developing such bodies. In no case have these spores
een seen to germinate."- Jones, L. R.; Giddings, N. J.; Lutman, B. F.; Investi-
ations of the Potato Fungus Phytophthora Infestans, Vermont Agricultural Experi-
ent Station Bulletin No. 168, pp. 84-86, August, 1912.

Over-wintering.- "It is clear from experiments that the mycelium of
hytophthora infestans spreads in the tissues of the potato tuber and finally
eaches the sprouts. The growth of the fungus is retarded when diseased tubers
re held in dry soil or at temperatures below 5°C. Infected tubers rot rapidly
hen placed in warm wet soil. This explains the wide variation in stand obtained
y earlier writers. A temperature of 23° to 27° C. and a well-watered soil were
ound to be the most favorable for the mycelium to spread in the tuber and grow
ut into the sprouts, both when partially and when wholly covered with soil.
nder these conditions the sprouts may become infected from 4 to 20 days after
lanting, regardless of their size and age. The time required is doubtless in-
luenced by the proximity of the mycelium to the buds and the external conditions.

"The mycelium of P. infestans may remain alive in seed tubers planted in the
oil for at least 45 days, and it is very possible that under conditions less
avorable for the soft rots which follow P. infestans in the tuber the fungus may
ive much longer. None of the author's results or observations tend to show that
he potato fungus is latent in the stems and leaves of plants growing from diseased
ubers, as stated by Massee.

"Laboratory tests showed that the fungus infects not only the sprouts but
lso the shoots that break through the soil. The mycelium grows from the tuber
nto the stem, where it travels up to the surface of the soil and sporulates, as
eld by De Bary and Jensen. This usually takes place in the small dwarfed shoots
n a hill.

"Potato tubers infected with P. infestans used for seed purposes and planted
nder field conditions may cause the development of an epidemic. The mycelium
grows from the parent tuber up into the stem exactly as shown in the laboratory
experiments. It later sporulates and foliage infection results. Ten such cases

were found and followed in northern Maine during the growing seasons of 1913 and 1914. All except two of these became centers for foliage infection, and severe epidemics of P. infestans followed.

"Conidia of P. infestans may be borne on the cut surfaces and sprouts of tubers when planted under field conditions. As the cut surface corks over or the tuber decays, the fructification of the fungus decreases. Spores taken from tubers two or three weeks after they were first planted showed only limited germinating capacity. No evidence was obtained tending to show that the conidia borne in the soil are instrumental in starting foliage infection.

"The mycelium of P. infestans spreads most rapidly in the cortical tissues of the stem, where it travels up more rapidly than down.

"Epidemics of late-blight may start from a single shoot or hill naturally or artificially infected with P. infestans. The infection spreads radially from the initial point of infection during the early stages of the development of an epidemic. These spots of infection in the fields probably come into existence through the planting of seed potatoes infected with P. infestans."- Melhus, I. E., Hibernation of Phytophthora Infestans of the Irish Potato. Jour. Agr. Research. 5: 99-100. October 11, 1915.

"Control.- Studies looking toward the prevention of the potato blight were begun during the middle of the past century. At first the greatest success was accomplished only in securing comparatively resistant sorts. Soon after the discovery of Bordeaux mixture, and more than thirty years ago, this fungicide was effectively used as a preventive of the late blight. By repeated experiments under a variety of conditions it has now been abundantly shown that the proper use of Bordeaux mixture will, in ordinary seasons, hold this disease in check, reducing its ravages to a small minimum. It is ordinarily advisable to begin spraying with 5-5-50 Bordeaux when the plants are about six inches high, and at least three thorough applications from ten days to two weeks apart are advised. In some cases two additional applications may be necessary. Again, it should be remembered that the fungus is carried over winter largely, or perhaps entirely, by a hibernating mycelium in the tubers, and that every effort should be used to secure seed potatoes from a field in which no blight or rot has occurred. If this latter could be done in connection with a system of rotation, there is no apparent reason why the disease might not be practically stamped out in any considerable region."- Duggar, Benjamin Minge. Fungous Diseases of Plants. Page 170-171.

Summary.
(From Vermont Bulletin 168)

"1. The potato disease termed the late-blight and rot has been known as the most serious of all potato diseases in Europe and America since about 1845, when its outbreak was the immediate cause of the Irish famine. In the State of New York alone a loss of 20,000,000 bushels in one year was attributed to this disease, and the percentage of loss may be even greater elsewhere.

"2. The disease does not attack the leaves, as a rule, until after the lossoming period, i.e., in late summer; if present and weather conditions favor, t quickly causes late-blight, which kills the foliage and thence passes to the ubers, causing the dry-rot.

"3. The disease is common in the Northeastern States, being favored by ather cool, moist summers. Farther south and west it is less common, probably nly occurring locally when introduced with seed from the North. It does not ong persist where the late summers are warm and dry.

"4. It is liable to confusion with such leaf diseases as the early-blight Alternaria) and leaf-blotch (Cercospora) and with various types of tuber rot.....

"5. The fungus develops first on the foliage, from which it passes by means f spores that are washed into the soil to the tubers, in which it hibernates.

"6. Jensen showed the possible efficacy of two remedial measures:

(a) Burying the tubers to a sufficient depth (about 4 to 5 inches) with soil to prevent the infection;
(b) Disinfecting tubers designed for seed purposes by exposure to dry heat, 40° C. (104° F.) for four hours. Neither of these methods has become established in practice.

"7. Studies of infection, dissemination, and disease control have shown:

(a) Tuber infection in the field may be prevented by spraying the soil even when the fungus is allowed to develop unchecked on the foliage. This is explainable only on the assumption that the primary tuber infection comes from spores washed through the soil.
(b) Tubers may also be infected from contract with blighting foliage at digging time.
(c) Secondary infection of tubers may occur either in the soil before digging or in the storage bin from spores developed on the surface of earlier infected tubers.
(d) When the tops are attacked by late-blight the harvesting of the tubers should be delayed until a week or more after the death of the tops. Longer delay does no harm unless the season be wet and the soil exceptionally heavy.
(e) Dry, cool storage is of primary importance, the use of lime or formalin disinfection for the tubers being valueless.
(f) Wind and water are probably the important agencies in local spore distribution, but leaf-eating insects also function and may carry the spores longer distances.
(g) Spraying the foliage with Bordeaux mixture has proved an almost complete remedy against both the Phytophthora blight and the rot, and also operates beneficially to the potato plant in other ways. Spraying experiments with this mixture have been made annually at the Vermont experiment station for 20 years, 1891 to 1910, on late or main-crop potatoes, three applications generally being made. The results were an increased yield in every case, ranging from 18 to 215 per cent. The average of the yields of the 20 years on the

sprayed areas was 268 bushels per acre as compared with 163 bushels on the unsprayed, a gain of 105 bushels per acre, or 64 per cent...."
- Jones, L. R., N. J. Giddings and B. F. Lutman. Investigatons of the potato fungus, Phytophthora infestans. Vermont Exp. Sta. Bul. 168. August, 1912.

Review

Describe the symptoms of late blight of potato on all parts of host attacked. At what season and under what conditions is an outbreak of this disease to be expected? Describe the life history of the fungus causing this disease. How is the disease carried over from year to year? How disseminated? How does spore germination and infection take place? Discuss control measures.

References.

(1) Duggar, B. M. Fungous diseases of Plants. p. 165.

(2) Stevens & Hall. Diseases of Economic Plants. p. 263.

(3) Taubenhaus, J. J. Diseases of Truck Crops. p. 322.

(4) Jones, L. R., Giddings, N. J. and Lutman, B. F. Investigations of the potato fungus, Phytophthora infestans. Vt. Exp. Sta. Bul. 168, August, 1912.

(5) Barrus, M. F. Late Blight and Rot of Potatoes. Cornell Exp. Sta. Cir. 19. May, 1913.

(6) Stewart, F. C. The persistence of the potato late blight fungus in the soil. Geneva Exp. Sta. Bul. 367. October, 1913.

(7) Melhus, I. E. Hibernation of Phytophthora infestans of the Irish Potato. Jour. Agr. Research. 5: 71, October 11, 1915.

(8) Morse, W. J. Two epidemics of potato blight and rot. Me. Exp. Sta. Bul. 169. November, 1909.

(9) Martin, W. H. Late blight of potatoes and the weather. New Jersey Exp. Sta. Bul. 384, 1923.

DOWNY MILDEW OF LETTUCE

Caused by Bremia lactuacae. Regel

1. Examine dried lettuce leaves showing the effect of the disease and also the downy growth of fruiting structures on the surface of the leaf. Sketch.

2. Mount conidiophores and conidia in water on a slide. Spread them as much as possible on the slide under the cover glass. Draw one or two conidiophores

n detail showing the exact character of the tips and the manner in which the pores are borne. Conidia germinate directly by means of a germ tube.

3. Write notes comparing this disease in all details with the late blight isease of potato.

References.

(1) Duggar, B. M. Fungous Diseases of Plants. p. 164.

(2) Bailey, F. D. Downy Mildew of lettuce. Ore. Exp. Sta. Biennial Crop Pest and Horticultural Report, 1911-12. p. 275.

(3) Anonymous. Plant diseases in forcing house and garden. Ohio Exp. Sta. Bul. 73. p. 226. 1896.

DOWNY MILDEW OR BLIGHT OF ONION

Caused by Peronospora schleideni Ung.

This disease has been variously known as onion mildew, blight or mold for more than half a century. It sometimes becomes quite serious in localities where onions are grown upon a commercial scale.

1. Laboratory

a. Symptoms.- Examine dry material and material preserved in liquid preservative and take notes on the appearance of diseased plants. Note the fruiting stage of the fungus on surface of leaves.

b. Life history

(1) Asexual stage.- Mount some of the conidiophores or examine a mount already prepared for you. If sections of leaves are available, note the conidiophores protruding from the stomata. Draw. Be careful to show exact shape and manner of branching of conidiophores. What is the shape of the conidia and where are they borne. Conidia germinate directly by a lateral germ tube. Compare with late blight of potato and lettuce mildew.

(2) Sexual stage.- This fungus also reproduces by means of sexually formed spores, oospores which are formed on the mycelium imbedded within the diseased tissue. These are large round, brown spores. Find some of them in the prepared sections of onion leaf and draw.

c. Notes.- From what you have learned in the laboratory and from a study of the references write complete notes on this disease.

Text discussion.

The main facts concerning this onion disease are briefly stated in the following extract from the Oregon Agricultural College Crop Pest Report.

"... This Disease is one that has long been known as a serious disease in England and Europe and other parts of the world as well as in various sections of the United States.... It has been reported from various sections of Western Oregon and has been particularly serious during the unusually moist season of 1912. It is found most commonly in Oregon on beaverdam soils.

"Symptoms.- The disease attacks the leaves, causing them to collapse. It usually appears first on a few plants in the field, but spreads rapidly in warm, damp weather. In the first stages of attacks the leaves of an infected plant will show a peculiar violet tint. If these leaves are examined closely this color is found to be due to the presence of a downy growth on the affected surfaces. In a day or two the leaves become weak and gradually collapse. The collapsed leaves dry up and may become covered with black mould, which the grower frequently mistakes for the cause of the trouble. It takes only three to five days from the first appearance of the trouble for the plants to be completely collapsed.

"Cause.- The disease is caused by a fungus belonging to a group of disease-producing forms commonly referred to as the "downy mildews" on account of the appearance of the fungus on the surface of the diseased parts..... This fungus, like many others, has two spore stages. In the summer stage the spores are formed on branches of the fungus body which appear in great numbers on the surface of affected leaves. It is these spore-bearing structures which cause the downy appearance mentioned above. The spores are spread by the wind and cause new infections. Since the time required for the fungus to kill the leaves is very short, it is readily seen why the disease spreads so rapidly over a field once it becomes started. The main part of the fungus body (mycelium) is within the leaf. This consists of very much branched thread-like structures which send short branches into the cells of the onion leaves. These absorb the sap from the cells and hinder the proper discharge of their normal functions. The result is that the leaves gradually become weakened and die. The loss of foliage results in decreased size of bottoms and consequent reduction in yield.

"On the mycelium within the tissues of the leaf, there are formed spores entirely different in character from those borne on the surface of the leaves. These spores may be referred to, for convenience, as resting spores. They serve to carry the fungus over the winter. They are abundant in the dead leaves or parts of leaves that lie on the ground over winter. In the spring, under favorable conditions, these spores grow and start the disease, which spreads rapidly in the field on account of the abundant production of summer spores on the surface of the leaves.

"Treatment.- In those sections where onion mildew is troublesome, the foliage should be kept covered with a fungicide during the growing season to prevent the germination of the spores on the surface of the plant and the consequent infection. The best fungicide for this purpose is Bordeaux mixture. It should be used in the 5-5-50 strength and should be applied at intervals of from ten days to two

eeks beginning when the plants show three leaves. Onion foliage is so smooth hat Bordeaux prepared in the usual way does not adhere readily. It is necessary, herefore, to add some sticker to the mixture."- Bailey, F. D. Oregon Agricultural ollege Biennial Crop Pest and Horticultural Report. 1911-12. pp. 239-240.

References.

1) Duggar, B. M. Fungous Diseases of plants. p. 162.

2) Whetzel, H. H. Onion blight. Cornell Exp. Sta. Bul. 218. p. 138. 1904.

3) Bailey, F. D. Onion mildew. Ore. Exp. Sta. Crop Pest & Hort. Report 1911 - 12. p. 276.

DOWNY MILDEW OF GRAPE

Caused by Plasmopara viticola (B & C.) Berl. and de Toni.

This is a widespread disease of grapes in certain parts of the United States and was introduced into Europe from this country. There it became a very serious pest in some of the grape growing regions. It was in connection with this disease that the fungicide known as Bordeaux mixture was first discovered.

1. Laboratory.

a. Symptoms.- Examine leaves and fruits affected with the disease and draw. Describe the signs of the disease.

b. Life History.-

(1) Asexual.- Make a mount by scraping a little of the downy growth from a diseased leaf and mounting it on a slide in water. Carefully draw a conidiophore to show manner of branching. Are conidia present in the mount?

(2) Sexual.- This fungus produces oospores somewhat similar to those of the onion mildew imbedded in the tissues of diseased leaves. If material is available examine and draw.

c. Notes.- Write an account of this disease from what you have seen in the laboratory and from a reading of the references.

2. Recitation.- Be ready at the next recitation to discuss any of the "downy mildew" diseases studied

Text discussion.

Hesler and Whetzel (3) give the following brief account of this disease:

"This disease was first observed in America in 1834. Some time prior to 1878 the pathogene was carried to Europe on America stock, for in September of that year it was first recorded in France. During the five preceding years, French growers had been warned against such importations from the United States on account of the downy-mildew of grapes. At that time European vineyardists were eager to use the American root stock on which to graft French vines as a remedy for the grape phylloxera, another pest of American origin which had already been so destructive to French viticulture. But in spite of the admonitions of the French scientists against importations from America, the downy-mildew finally reached the European vineyards. From France the mildew-pathogene spread throughout Europe, where it is now a very troublesome foe. In the United States the disease is very generally known, being more destructive to European varieties than to domestic forms. No variety is notably resistant under all conditions. Both smooth and pubescent, wild and cultivated, sorts are affected. The downy-mildew also occurs on five-leaved ivy, a close relative of the grape.

The greatest losses to American viticulturists from this disease are incurred in northern United States, where in some localities it has been estimated that 25 to 75 per cent of the crop is destroyed. Losses in Europe have been enormous. The disease is destructive not only to the berries but to practically all young or green portions of the vine. The nature of the losses is as follows: (1) shelling; rotting and mummification of the fruit; (2) spotting of the foliage, often resulting in premature defoliation; and (3) sometimes a dwarfing and killing of canes and leaves.

"Symptoms.- On the upper surface of the leaf the first signs of downy-mildew are in the form of small greenish yellow indefinite spots, the margins of which gradually merge into the darker green of the leaf. In a short time there appears within the spot a network of small, reddish brown lines, the discolored smaller veins. These lines become more pronounced until the whole diseased portion is brown, dry and brittle, and eventually cracked. These symptoms apply particularly to susceptible leaves. On the foliage of the more resistant vines there occurs only a slight change in color; within this area are found numerous brown punctations, which do not unite to form a dead, brown spot in the leaf-tissue. The disease on the lower surface of the leaf is at first similar to that on the upper, but is very soon covered by a downy, white growth which is most pronounced just beneath the greenish yellow portion mentioned above. On the brown portion the whitish felt, the mildew-pathogene, soon disappears. The amount of mildew on a leaf depends on weather conditions and on the variety. The lower portion of a vine may be denuded of its foliage.

"The canes, leaf-petioles and tendrils are subject to attack, especially in wild varieties. At first the affected portion has a water-soaked shiny appearance, which is attended by a local swelling of the tissue. The lesion is bare at first, but soon the mildew covers it. In older cases the affected area becomes brown and dead, and a depression results. In cases of severe attack the cane is dwarfed, the leaves remain small, and the cane may die.

"The fruit and flowers of wild varieties commonly show the downy-mildew. The attack may come at any time from flowering to maturation. The first sign of the trouble on the fruit is a hardening of the berry, together with a change from its normal color to a grayish blue lead-color. It is during this state that the mildew appears. In later stages the berry withers, turns brown or red, and finally shrivels into a mummy. Diseased fruits shell very easily.

"Cause.- The downy-mildew pathogene is a fungus, Plasmopara Viticola. It has both a sexual and an asexual stage, the former represented by oospores, the latter by conidia. It is generally agreed that the oospores carry the fungus through the winter in the old fallen leaves. In the spring these oospores germinate, forming a conidophore, on which is borne another spore, a conidium. The conidium in turn, at maturity, germinates, but instead of forming a germtube, its contents break up into six or eight extremely small naked spores. Each of these has two cilia which give it the power of motion; on account of this motile character these small naked bodies have been named swarmspores. The production of swarmspores, which really initiate the disease, begins in February and continues until June. They are formed and discharged within 24 hours. The swarmspores or their parent spores, the conidia, are spattered in drops of water or mud by heavy rains to the leaves of the vine. After reaching the leaf, the swarmspore, which is still a naked protoplasmic mass, soon comes to rest and surrounds itself with a thin wall. Then a germtube is developed which penetrates the leaf by way of a stoma, always on the lower side of the leaf..... The germtube within the tissues develops into mycelium which brings about the various changes in the affected parts as evidenced by the symptoms already described. Within a short time the fungus masses itself beneath a stoma, through which opening several (four to ten) conidophores pass. These grow from most of the stomata in the infected region, each conidiophore reaching a height of about one-fiftieth of an inch. In mass they present a whitish, downy appearance - the downy-mildew. These structures form branches, on the ultimate tips of which are borne conidia. The conidia are blown to other susceptible parts of neighboring vines, where new spots are formed. In the autumn, affected leaves fall to the ground, carrying the fungus within them. Before winter arrives oospores, as many as 200 to the square centimeter, are formed. In the spring the oospores are set free by a rotting of the leaves, germination occurring in the soil.

"A temperature of 77° to 82.4° Fahr. is best for the growth of the fungus. Shaded, moist situations are favorable for its development. Long, dry periods do not kill the pathogene, although such conditions seriously check it. Heavy rains of short duration followed by sunshine and winds are not favorable to the spread of the fungus. Most injury is done during hot, wet weather.

"Control.- Since the fungus hibernates in old fallen leaves it is commonly recommended that these be plowed under early in the spring. The real value of such an operation, however, is not definitely known. The vines should be sprayed to protect them against attacks of the downy-mildew pathogene in any case. Use bordeaux mixture 5-5-50, making five or six applications during the season. The first should be made just before the blossom buds open; the others at intervals of two weeks."- Hesler, Lex R. and Herbert Hice Whetzel. Manual of Fruit Diseases. pp. 237-242.

References.

(1) Duggar, B. M. Fungous Diseases of Plants (Text). p. 152.

(2) Gregory, C. T. Spore germination and infection with Plasmopara viticola. Phytopathology, 2: 235, December, 1912.

(3) Hesler & Whetzel. Manual of Fruit Diseases, p. 237.

SUPPLEMENTARY LIST OF DOWNY MILDEWS

The preceding four exercises are studies of representative species of four genera of downy mildews. If time permits there are many other species of some of these genera and also other genera which might profitably be studied.

Following is a short list of additional species:

Peronospora parasitica (Pers.) DeBary. On cabbage, cauliflower, radish, etc.

Peronospora effusa (Grev.) Rab. On spinach and other members of the same family.

Peronospora trifoliorum deBary. On clover and alfalfa.

Peronoplasmopara cubensis (B. & C.) Cl. On cucumbers and other cucurbits.

Sclerospora philippinensis Weston. On maize in the Philippines.

CHAPTER XIX.

Diseases caused by fungi - Ascomycetes.

Fungi belonging to the class, Ascomycetes, commonly known as "sac fungi", are responsible for some of our most serious plant diseases. Examples of diseases caused by this class of fungi are: peach leaf-curl, brown rot of stone fruits, stem rot of clover and alfalfa, Northwestern apple anthracnose, the powdery mildews, ergot, black rot canker of pomaceous fruits, European canker and apple scab.

Most ascomycetes have both a conidial, or asexual stage and a perfect, or ascigerous reproductive stage. In many cases, however, only the conidial stage occurs in the most harmful or parasitic phase of the fungus while the perfect stage is produced saprophytically on dead parts of the host, as, for example, apple scab.

PEACH LEAF CURL

Caused by Exoascus deformans. (Berk.) Fckl.

This disease is characterized by distortion of the leaves, the young shoots and sometimes the fruits of the host plant.

1. Laboratory Study.

a. Symptoms.- Examine diseased leaves, twigs and fruits of peach and make sketches to represent conditions shown by these specimens. Describe symptoms in notes.

b. Life History.- Examine, with the microscope, sections of diseased leaves. Find the layer of sacs (asci) breaking through the cuticle at the surface of the leaf. Try to determine in just what part of the leaf tissue, these sacs are formed. How many spores in a sac? Can you find any fungus threads (hyphae) penetrating the leaf tissue beneath the epidermis? Compare the section of diseased leaf with section of normal leaf. Can you detect any difference? If so, what? Draw a narrow section across the diseased leaf to show the layer of sacs and their relation to the host leaf tissue. From text and other references find out what is known concerning perpetuation and dissemination of spores and infection of host.

2. Field Study.

If season and weather conditions permit, a very profitable field trip may be made to study this disease in the orchard. Any or all of the following points may be observed depending upon the time of year and other conditions.

a. Deformed and dead shoots killed previous season.
b. Dead leaves killed last season and still clinging to tree.

c. First signs of infection on young leaves soon after they burst from the bud.
d. Watch development of infected leaves to maturity (requires repeated trips.)
e. Observe results of control measures.
f. Estimate percentages of diseased leaves on sprayed and on unsprayed trees.

3. Notes.- From what you have learned in laboratory and field and from reading the references cited below, give a detailed write-up of this disease.

Discussion.

Hesler and Whetzel (2) give the following account of the peach leaf-curl disease.

"The Disease has long been known in England, where it was described as early as 1821 by an English gardener as blister. It has become widely distributed throughout the world wherever peaches are grown, being most serious near the seacoast or in the region of large interior lakes. In the United States and Canada it is very common and destructive in the peach-growing sections about the Great Lakes. Likewise in the Pacific Northwest it has been very troublesome in regions of considerable annual rainfall.

"Peach leaf-curl is regarded as the most serious fungous disease affecting this fruit in cooler climates. In warmer climates, on the other hand, brown-rot assumes this role. The fruit being rarely attacked by the leaf curl pathogene, the losses involved are usually underestimated. The annual toll is said to be three millions of dollars. The character of the losses is:

(1) Loss of leaves in the spring, followed by a forcing of a new crop of foliage later that year, which lowers the vitality of the tree;
(2) Partial or total failure of trees to set or hold a crop because of defoliation;
(3) Repeated loss of leaves for several seasons in succession, resulting in the death of the trees;
(4) Injury to trees by killing the twigs;
(5) Stunting of nursery stock due to the death of the shoot from the bud; curl-affected nursery buds never make good trees.

"Symptoms.- Most peach-growers are familiar with the symptoms of peach leaf-curl, particularly in its later stages. The first evidence of the disease, however, may be frequently over-looked. In the spring shortly after the leaves begin to unfold there is a puffing and folding of these organs. The leaf-blade becomes thickened and puckered along the midrib, causing the leaf to curl. The diseased portion becomes yellowish, with tints of red. The leaf is thickened, becomes brittle, and finally shows a characteristic silvery bloom over the upper surface. Curling may be confined to a part of the blade, or the petiole, or may involve the whole leaf. Affected leaves finally die and drop from the tree, in some cases the entire tree being defoliated. A new set of leaves is then formed

om the dormant buds following defoliation. Affected twigs show a marked swelling nd are stunted in length. Their color changes to pale-green and yellow. While ne hypertrophy involves the current year's growth for the most part, sometimes ne pathogene extends down the side of the previous season's growth, forming a vollen ridge. Badly infected twigs are usually killed, but the growing tip may evelop a healthy shoot, leaving a swollen canker-like lesion at its base. An xudation of gum often accompanies the lesions on the twigs. The flowers and oung fruits are often attacked, but because they soon drop away this symptom of url is seldom observed. In general, the sickly yellow, curled foliage, and the inal defoliation of the trees the latter part of June, followed by a refoliation, re the most striking symptoms of this disease.

"Cause.- The peach leaf-curl disease is caused by the fungus Exoascus eformans, so named because it forms its asci on the outside of the host, and ecause it deforms the leaves. The mycelium of the pathogene grows between the ells of the leaf, stimulating them to abnormal increase in size and number, and obbing the leaf of its green chlorophyl. When the spores are to be produced, he mycelium invades the cuticle of the leaf, and at once gives rise to sacs (asci) hich bear from 3 to 8 ascospores. The presence of the asci on the upper surface f the leaf gives the leaf its silvery appearance. At maturity, the ascospores ud extensively within the ascus, giving rise to spores suggesting conidia. The scus then empties its contents through a crack at the top..... It is the pinion of most investigators that the fungus hibernates on the bud-scales in the orm of spores (kind unknown), and that these spores germinate during the spring ains at a time when the buds swell, and that the germtube penetrates the very oung leaf as it emerges from the bud. This opinion is based on the following ircumstantial evidence:

(1) Lesions appear on leaves just as they are protruding from the buds;
(2) Leaves from buds sprayed before such buds swell show little or no infection during the season, while unsprayed buds on the same tree curl badly;
(3) Buds sprayed after they swell, and especially after rains, show curled leaves;
(4) The disease occurs only during cold, wet springs.

"From the above data it may be said that, theoretically, some sort of spores re lodged by the wind or rains among the hairs of the bud-scales during the late ummer, and that these spores remain there dormant until conditions favorable to nfection arise the following spring.

"The effect of the environment upon this disease is very marked. This is oticed in a general way in connection with the geographical range of the disease, hich is most common and severe in the neighborhood of large bodies of water. he combination of conditions most favorable to an epiphytotic of peach leaf-curl is a cold wet-period following warm spring weather. Warm weather starts the buds; a cold wet spell immediately following results in a saturation of the leaf-tissue vith water, due to a lowering of the temperature and to the high humidity of the atmosphere. The buds are retarded, their cells gorged with water, and their walls distended, while the damp atmosphere permits spore-germination and infection. As already noted, curl is most severe near large bodies of water; this is doubtless due to the increased humidity of the air in these localities, and to reduced temperature in the early spring as evidenced by the more frequent occurrence of fogs

n such regions. Heavy dews can exert but little influence on curl, since the oisture and temperature factors are not sufficiently pronounced nor of adequate uration to effect a response on the part of the host and the parasite. Rainfall eems to have considerable influence on the disease. In the Pacific Northwest, or example, little or no curl exists east of the Cascade Mountains, where the nnual rainfall is light; on the other hand, west of these mountains the rainfall s heavy and curl is very destructive. In these two regions the temperature is pproximately the same.

"Control.- The time and thoroughness of the application of remedial measures re important points in the control of peach leaf-curl. It is imperative that such ork be done before the buds swell in the spring. Conjectures with reference to he winter and spring activities of the pathogene lead one to infer that spraying hould prevent the disease. It has been shown that the application of almost any ommon fungicide ordinarily controls the trouble satisfactorily. One spraying is ufficient and may be made in the fall, or in the spring before the buds swell. ever spray more than once, as it is a waste of time and materials; and do not pray for curl after the leaves are expanded, for such an operation is useless and angerous. Spray thoroughly, making sure to coat every bud. If the season is avorable to curl, the unsprayed buds will show the disease. Ordinarily lime-ulfur solution of standard Baume test, 32°, at a strength of 1 to 15 or 1 to 20, s effective. If San Jose scale must be combatted, both troubles may be controlled y using the fungicide at scale strength, 1 to 8. Scale can be controlled as ffectively by spraying in the fall as in the spring. Bordeaux mixture of any trength, or copper sulfate 2 pounds to 50 gallons of water, are also effective in ontrolling peach leaf-curl, and may be used when scale is not a factor to be con-idered."- Hesler, Lex R. and Herbert Hice Whetzel. Manual of Fruit Diseases. ages 277-282.

Review.

Describe the symptoms of peach leaf curl and give the life history of the ausal organism (Exoascus deformans). Discuss the relation of hold-over charac-eristics and the time and point of infection to control measures. Give the time imits within which spraying for peach leaf curl must be done in order to be ffective. Are these time limits calendar dates? Why? Name susceptible and esistant varieties of peach to leaf curl.

References.

1) Duggar, B. M. Fungous Diseases of Plants, page 176.
" " " Peach leaf curl. Cornell Exp. Sta. Bul. 164, page 371. 1899.

2) Hesler & Whetzel. Manual of Fruit Diseases, page 277.

3) Pierce, N. B. Peach leaf curl; Its nature and treatment. Div. Veg. Phys. & Path., U.S.D.A. Bul. 20. 1900.

4) Stevens & Hall. Diseases of Economic Plants (Text) page 126.

) Massee, Geo. Diseases of Cultivated Plants and Trees (Text), page 138.

) Jackson, H. S. Peach leaf curl. Ore. Exp. Sta. Crop Pest Report 1911 - 12. page 253.

) Wallace & Whetzel. Peach leaf curl. Cornell Exp. Sta. Bul. 276: 155. 1910.

) Reddick, D. Fall Spraying for peach leaf curl. Cornell Exp. Sta. Cir. 31: 65. 1915.

) Mix, A. J. Biological and cultural studies of Exoascus deformans, Phytopathology 14: 217. May, 1924.

OTHER DISEASES CAUSED BY EXOASCUS.

There are two other well-known diseases caused by species of Exoascus, namely, itches' broom of cherry and plum pockets or bladders. Neither of these diseases s usually of as much economic importance as peach leaf-curl. If time permits they ay be studied briefly in comparison with leaf-curl. Brief outlines for these two iseases follow.

WITCHES' BROOM OF CHERRY

Caused by Exoascus cerasi. (Fckl.) Sadeb.

This disease causes a curling of the leaves somewhat similar to peach leaf url but in addition it causes a bushy growth of twigs known as witches' broom. his characteristic has given the disease its common name.

1. Examine diseased leaves and compare with peach leaf curl. Also examine hotographs of witches' broom. Read references and write notes comparing this isease and its causal organism in all details with peach leaf curl.

Discussion.

Hesler and Whetzel (2), in their Manual of Fruit Diseases, give the following hort account of witches'-broom.

"Witches'-broom is a peculiar type of gall in which there is an over-produc- ion of whole organs, resulting in a brcom, or nest-like habit of growth. This ame is in general use in North America. In England, the disease is called, hunder-bushes, bull-boughs, bull-wood and bull-branch. The Germans refer to it nder the name Hexenbesen, while the French call it Balai du Sorciere.

"The disease is very common and destructive in Europe, where both sweet and our cherries are affected. Although the English sweet cherry (Prunus avium) is ommonly cultivated in eastern United States the disease was not recorded until 886, when it was reported from Germantown, Pennsylvania. About ten years later was found in Long Island at scattering points. The witches'-broom disease of herry is not a disease well-known to American cherry-growers. It occurs sparing- in New York, but is said to be common in Oregon. It has also been observed in few other states, including Washington, Minnesota, Pennsylvania and New Jersey. he disease is of most interest perhaps because of the fact that the pathogene preads so slowly, and on account of the peculiar effect on the cherry. Its story thus far in this country does not indicate that the disease should ever be eared by cherry-growers. Records show that the disease may affect in this country he following cherries: English sweet cherry (Prunus avium), sour cherry (Prunus erasus), wild black cherry (Prunus serotina), wild red cherry (Prunus pennsylvan- ca), choke-cherry (Prunus virginiana). Several varieties and species of plums re also affected.

"Symptoms.- The twigs and leaves are susceptible to this disease. On the ormer it produces a very striking deformity, although not a killing, of the ffected parts. Infected branches become more numerous and are more or less longated. Authorities disagree on the matter of whether affected twigs are hickened. In some cases the twigs become so numerous that they are not able to ear their own weight, and as a result the abnormal portion of the tree droops. n such cases the tips of the branches usually turn upward. The over-production f branches at a local region results in a broom-like growth. Some of the brooms re large enough and so conspicuous that they are very noticeable from a distance. his is especially true when the tree is bare of leaves. These broom-like growths re also conspicuous at blossoming-time; for they bear few or no flowers, hence he affected portion stands out in bold contrast to the remaining blossoming- ranches. Leaves also come out on brooms before they do on healthy branches.

"Affected leaves take on a crinkled shape and a reddish discoloration. The isease on leaves is usually referred to as leaf-curl, and the abnormalities ex- ibited are very much like those of the peach leaf-curl. Affected leaves fall rematurely and later new foliage appears. On their lower surfaces a whitish oat may be found prior to defoliation.

"Cause.- The causal fungus, Exoascus cerasi, is very closely related to the ungus causing peach leaf-curl and to that of plum-pockets. Its full life-history s not known. The mycelium invades the twigs, living in them from year to year. oth the bark and wood are attacked. The pith-cells, medullary-ray cells and uter cells of the bark (hypodermis) are greatly increased in number as a result f stimulation set up by the parasite. The sclerenchyma fibers, on the other hand, re fewer or lacking. The stimulating effect of the fungus brings about a condi- ion whereby, instead of flower-buds being produced as in ordinary cases, abnormal wigs are developed, giving the broomy aspect already described. The mycelium nvades the leaves, causing them to curl and fall. Finally, before defoliation, he fungus forms a fruiting layer of asci with ascospores on the lower surface of he leaves. These bodies give to this surface the whitish appearance already entioned.

"Control.- Fortunately the disease is not usually of sufficient importance to require urgent attention. Little experimental work has been done on which to base recommendations for American conditions. Since the brooms bear neither flowers nor fruit and are a source of trouble, they should therefore be removed. The cut should be made several inches below the lowest point in the diseased portion. It is said that this method readily controls the disease."- Hesler, Lex R. and Whetzel. Manual of Fruit Diseases. pp. 188-190.

Review.

Describe this disease, give life history, and compare with peach leaf curl and the disease known as plum pockets which follows this exercise.

References.

(1) Duggar, B. M. Fungous Diseases of Plants, p. 185.

(2) Hesler & Whetzel. Manual of Fruit Diseases, p. 188.

(3) Stewart, F. C. Witches' broom of cherry trees. N. Y. (Geneva) Exp. Sta. Report 14: 532. 1895.

(4) Stevens & Hall. Diseases of Economic Plants, p. 119.

(5) Massee, Geo. Diseases of Cultivated Plants and Trees. p. 139.

(6) Stewart, F. C. Notes on N. Y. plant diseases. N. Y. (Geneva) Exp. Sta. Bul. 328: 340. 1910.

(7) Lawrence, W. H. Witches' broom of cherry. Washington Exp. Sta. Bul. 83: 25. 1907.

PLUM POCKETS

Caused by Exoascus pruni. Fckl.

1. Examine plums attacked by this disease and also photographs or illustrations in texts. Make habit sketch. Read the following references and write notes.

References.

(1) Duggar, B. M. Fungous Diseases of Plants, page 183.

(2) Hesler & Whetzel. Manual of Fruit Diseases, p. 373.

3) Stevens & Hall. Diseases of Economic Plants, p. 141.

4) Hesler, Lex R. Plum Pockets. N. Y. Agr. Dep't Bul. 79, page 1198. 1916.

5) Swingle, D. B. and H. E. Morris. Plum pocket and leaf gall on Americana plums. Montana Exp. Sta. Bul. 123. February, 1918.

Review.

Name three diseases of stone fruit trees caused by three different species of Exoascus. Compare these three diseases, mentioning likenesses and differences.

BROWN ROT OF STONE FRUITS

Caused by Sclerotinia cinerea. (Bon.) Schrot.

This disease attacks all kinds of stone fruits and is not uncommon on pomaceous fruits such as apple and pear. Under suitable weather conditions for the growth of the fungus, enormous damage sometimes results to stone fruits, especially to the peach. Prunes and cherries also sometimes suffer severely.

1. Laboratory Study.

a. Symptoms.

(1) On Fruits.- Examine diseased fruits of cherry, prune, peach and apple. Note the type of rot in the flesh and also the tufts of conidiophores and conidia appearing on the surface of the fruits. These conidia are the summer fruiting stage, also known as conidial stage and asexual stage. Now examine old mummified fruits which have lain on the ground for at least eighteen months. Note the condition of the fleshy part of the fruit. What has happened to it? Note also the cup-like fruiting bodies attached. These represent the perfect or ascigerous (ascus bearing) stage. Make drawings to represent all features observed.

(2) On twigs, spurs and blossoms.- Examine twigs, spurs and blossoms which have been attacked by the brown rot fungus. Note cankers on twigs, and also note spurs that have been killed and blossoms that have been blighted by the fungus. Make such sketches as will best show the effect of the disease on these parts of the tree. Look for conidial tufts on blighted spurs.

b. Morphology and life history of causal organism.

(1) Conidial or Imperfect Stage.- Mount conidiophores and conidia for examination under the microscope. Draw to show shape and manner of branching of conidiophore and conidia attached. What part does this stage play in the life cycle of the fungus?

(2) Perfect or ascigerous stage.- Examine longitudinal sections of cup (ascus-bearing fruiting body called apothecium). Note the main body of the cup and the lining layer (hymenium) made up of sacs (asci) and sterile threads (paraphyses). Draw. Now take a fragment of a cup, about half the size of a pin head, crush it in a drop of water on a slide, put on a cover glass and examine under the microscope. The sacs, containing eight spores each, will appear much more distinct than in the section of cup previously studied. Draw.

c. Notes.- Write up in detail everything you have learned about brown rot in the laboratory, then read the references and complete your notes.

2. Field Study.

If season and weather permit, make a study in the orchard, of as many of the following features as are available at the time:

a. Rotted fruits hanging on the tree and producing conidia.

b. Twig cankers, blighted spurs or blossoms. Look for conidial tufts on blighted parts.

c. Mummies on the ground producing cups.

Text discussion.

The following quotation from Hesler and Whetzel (2) applies especially to the peach but many of the statements may be applied to the disease on other fruits as well. Cherries and prunes frequently are damaged as much as peaches by this disease.

"This disease, which is called brown-rot of stone-fruits, mold, blossom-blight, twig-blight, peach-rot, brown-rot canker, and other names, was not given serious consideration in America prior to 1881. It is now a well-known fungous trouble wherever the peach is grown, both in Europe and in the United States. The pathogene causing brown-rot probably came from some foreign country. History shows that the disease has been more serious in some years than others in America. In 1891, 1895, and during subsequent years brown-rot has been of considerable importance in the Delaware and Chesapeake peninsula. Alabama growers experienced a severe epiphytotic in 1897, while in 1900 the disease was the most conspicuous and the most destructive in Georgia since the beginning of stone-fruit culture in that state.

"Brown-rot is most prevalent and most destructive in the warmer peach-growing states, such as have already been enumerated. In warm, wet seasons the trouble is severe in the northern states. The light-colored varieties are generally regarded as the most susceptible. Those showing least rot in Georgia are the Carman, Early Crawford and others. Observations on the question of varietal resistance need to be extended.

Losses.

"The average annual loss to the peach-growers of this
untry because of brown-rot is placed at $5,000,000. For the year 1900 Georgia
owers are said to have lost from $500,000 to $700,000, and the average annual
ss is estimated to be not less than 40 per cent. Again, in 1909 the loss of
aches due to brown-rot in Georgia, with only one-third of an average crop, is
id to have reached $1,000,000..... While in the northern states and in Canada
ach-growers do not experience any such calamities, yet these regions are by no
ans exempt from the disease, and in some years the outbreak is severe. The loss
om this disease is not limited to the fruit-growers, but is felt by the trans-
rtation companies, the commission men and the consumers. A shipment of fruit
ich shows great promise as it leaves the orchard may reach the market in a
rthless condition. The disease is destructive in one or more of the following
ys:

(1) Blossoms may be blighted, destroying the set of fruit;
(2) Twigs may be blighted, thus inflicting serious injury on the tree;
(3) Large limbs may be cankered, which form of the trouble is of great importance in parts of New York State;
(4) The crop of fruit itself may be partially or wholly destroyed as a result of attacks by the brown-rot pathogene; green fruits, if injured by insects or hail, may be rotted extensively; and likewise ripe fruits on the tree, in transit, or in market may be wholly ruined as a result of rotting.

"Symptoms.- The fruits are most commonly affected. However, other organs of
e host are also susceptible to the disease; these include the blossoms, twigs,
mbs and occasionally the leaves.

"In America the flowers are commonly affected, resulting in a serious blossom-
ight. When the blossoms are opened, the petals turn brown and shrivel, but do
t fall. Grayish tufts composed of the fruiting structures of the pathogene show
the affected blossoms. Twig-blight follows blossom-blight very closely, the
rmer being a direct result of the latter. The leaves on such twigs wither and
e and cling to the twig, as in the case of fire-blight of pears. Twig-blight
y also result from the spread of the pathogene from affected fruit into the twig
way of the fruit-pedicel. This happens very commonly in New York State
chards. From the twig the causal pathogene passes into the larger limb where it
preads out, forming a canker. The brown-rot canker is a definite dead area in
he bark, the surface is sunken, and the lesion is accompanied by a flow of gum.
he disease is said to affect peach leaves, showing itself as a shot-hole.

"Fruits generally show signs of brown-rot after they are half grown, the sus-
ptibility of individuals increasing as they approach maturity. The lesion on
he peach is at first evident as a small, more or less circular, dark-brown,
ecayed area with a rather indefinite line of demarcation between the healthy and
iseased portions. This rapidly enlarges, and soon the fruiting structures of the
thogene appear on the surface as grayish tufts. At first these tufts occur
paringly, but in a day or so the original rotted spot may become densely dotted
ith the characteristic ashen mold. With the enlargement of the lesion the whole
uit becomes involved, is brownish, shrunken, and eventually shrivels into a
ark mummy. The mummy may cling to the tree or it may fall to the ground. While
the tree mummies cling together in groups of two or more.

"Cause of brown-rot.- The pathogene, the fungus Sclerotinia cinerea, bernates in both the fallen and hanging mummies, and in the cankers. From lerotial crusts in the fallen mummies arise apothecia in the spring of the year. e hanging mummies furnish a habitation for conidia through the winter; in the ring these spores are carried to the susceptible parts. These old clinging mmies as well as the cankers also contain mycelium of the fungus, and in the ring conidia are developed from these sources. The source of the primary in- ulum is found, then, in both the fallen and hanging mummies, and in the cankers. e inoculum itself consists of both ascospores and conidia, of which the latter e the more important. In fact, it sometimes appears that ascospores are not veloped every year, and that they play a very minor role in the life-history of ie brown-rot fungus. Spores of either kind are carried to the blossoms, where lossom-blight is induced. The mycelium grows throughout the blossoms, enters the vigs, then finally passes into the large limbs. Conidial tufts develop on the etals, and even on the cankers and blighted twigs, if the atmosphere has a high elative humidity. The conidia from these sources start the disease in other parts f the peach. From one or more of the several possible sources conidia are carried o the half-grown fruits. These spores germinate, and the tubes enter through an njury; the curculio and the peach-scab fungus are the chief agents in making such ounds. The mycelium develops profusely within the tissues of the fruit, the esult showing externally as a brown-rot. Conidial tufts soon appear and conidia re liberated to continue the destructive action of the fungus. Where two or more eaches touch each other, the mycelium grows from the infected peach through the oint of contact to the other. In this way the fruits are made to cling to each ther, several in a group.

"The fungus is highly favored by a series of cloudy days accompanied by requent showers, especially at picking time; consequently a great amount of fruit ay be destroyed under such weather conditions. Prolonged drizzly weather is far ore dangerous than a heavy rain followed by clearing. Hot weather favors the apid growth of the fungus and increases the danger of its destroying the crop. n the other hand, in a dry, cool season the crop may be expected to remain rela- ively free from brown-rot."- Hesler and Whetzel. Manual of Fruit Diseases. 1917. p. 270-275.

Additional life-history facts.

Norton, Ezekial and Jehle (13) have recently published some very interesting ata on life history studies of Sclerotinia cinerea with especial reference to he perfect or apothecial stage. Some of their statements are quoted below.

"Historical.

"The apothecial stage of the common American fruit-rotting Sclerotinia was irst found by Norton in Maryland orchards in April, 1902. It was briefly described in Science (11) and more extendedly in the Transactions of the Academy of Science of St. Louis (12). Cultures were made at that time and reported in the above publications demonstrating the development of the common Monilia form from the ascospores of these newly found apothecia.....

"Jehle recorded the following observations in an orchard in Niagara County, New York. This orchard was very thoroughly cultivated during the summer of 1911 up to the trunks of the trees. Scarcely a single whole peach mummy could be found anywhere in this orchard in the summer of 1911 until after the peaches were picked. Practically no brown-rot developed on the fruit during that summer, but in picking the peaches, all of the unsound fruit was thrown to the ground. The peach mummies which resulted from the infection of these fruits by the brown-rot fungus remained on the ground during the winter and produced an abundant crop of apothecia the following spring. These apothecia grew from mummies which were still whole. This orchard was not plowed in the spring of 1912 until after the apothecia were found.

"Valleau (14) working with buried plum and apple mummies found that no apothecia developed until the second spring..... In 1918 Pollock (15) found that plum mummies ten years old were still capable of producing apothecia.

Wormald (16) first found Sclerotinia apothecia in England in 1920, and has published on strains, biology, and on diseases caused by Sclerotinia in England. Roberts (17) first reported the production of apothecia under natural conditions from mummies produced the previous summer.

Apothecia appear at blossoming time.

"The close correlation of the time of blooming of peach trees and the appearance of apothecia was noted by Norton (12) and has been frequently mentioned since then. Even when blooming is unusually early, as in 1921 for instance, the apothecia appear in greatest abundance at this time. In the year mentioned peaches in local orchards were in full bloom between March 19 and 23, and this was also the period of maturity of most of the apothecia.

In 1922 about 15% of the apothecia had matured by April 4, when peach blossoms were just opening. Six days later when blooming was finished all the apothecia had opened and many of them had already dried up.

"The earlier apothecia may come out while the peaches are still in the pink-bud stage. The majority mature while the peaches are in full bloom, but a few apothecia are usually to be found in good condition for a week or so after blooming is over.

"In New York Jehle observed that the apothecia began to appear when the early varieties began to flower and continued to appear until the late varieties were through blooming.

"Moisture and air relations.

"Lack of water appears to be the factor, in this region, that largely limits the growth of most of the apothecia..... As the season advances and the ground dries out the apothecia on mummies in exposed places also dry up so that only those sclerotia in specially favored locations will mature apothecia.

"As a rule, fully developed apothecia have been found in low, moist spots, uch as furrows and other depressions near the trees. As was observed by Dandeno he presence of a mulch of dead grass, matted leaves or other debris which will eep the mummies moist even when entirely above ground is quite favorable to pothecial development.

"In bare ground sclerotia partly buried in the soil may be the only ones able o produce apothecia. A mummy imbedded in even comparatively dry soil may be moist nough on the lower side to do this. Cunningham (18) observed that apothecia eveloped from mummies in compact soil, due to a better moisture supply than in reshly plowed land. A short period of drying is, however, not fatal to apoth- cia.....

"While an abundant though not necessarily constant supply of moisture is ssential to the development of apothecia, they will not grow if entirely sub- merged in water. Experiment also showed that apothecia would not grow in a ightly stoppered bottle though kept moist, while checks developed abundantly in similar bottle that was not stoppered.

Depth necessary to bury sclerotia to prevent apothecial production.

"Three bushels of peaches and plums were inoculated in July, 1920, allowed to rot and most of them placed in sandy soil under natural conditions, at five different depths in the soil: (1) on the surface, (2) half covered with soil, (3) entirely covered, (4) buried one inch, and (5) buried two inches. Apothecia were produced in March, 1921, more abundantly from the first three depths, none at all developing from the two-inch deep sclerotia and only a single apothecium growing from a mummy buried one inch.....

These experiments have then shown that (1) burying brown-rot mummies below the surface of the soil prevents the production of apothecia, the precise depth necessary doubtless varying with the type of soil, and (2) if such buried mummies are brought again to the surface of the soil, they are still able to produce apothecia the next spring."

Age of sclerotia and production of apothecia.

"Until recently it has been the general opinion that under natural conditions apothecia arose only from mummies that had been in the soil for at least two winters, that is, a fruit infected with brown-rot in the summer of 1920 would not be able to produce apothecia until the spring of 1922. As was shown above, this idea was unfounded.

"However conditions are apparently such in some, if not most, seasons that sclerotia one year old are not able to produce apothecia. Such for instance appeared to be the case in the spring of 1923 in the University orchards at College Park. The 1922 mummies here were unmistakable since there had been no peach crops the previous two years. Careful search failed to reveal the presence

of a single apothecium on the many 1922 mummies that appeared to be in favorable locations, while the occasional bits of old sclerotia left on the ground often bore apothecia.

"A similar case observed in 1902 and 1903 by Norton has been mentioned above. Brown-rot was common in 1901. Few apothecia were noted in 1902 (19) while as had been predicted apothecia appeared abundantly in 1903 (20).

"Observations in general indicate that apothecia appear more frequently on older mummies than on the fresh ones. The age of mummies is however often difficult to establish by observation.

"It has been possible (21) after refrigeration to produce apothecia from a plum mummy just twenty-five weeks after the date of inoculation. The greatest age at which mummies have been reported to produce apothecia is ten years (25)."

A closely related blossom and spur blight.

Barss (23) has recently described what is believed to be a distinct species of fungus which is very closely related to the conidial stage of the common brown-rot fungus. A brief quotation from his bulletin will give the chief facts as known about this species.

"The true brown-rot disease which is so widespread in the United States is caused by a fungus usually designated by the scientific name Sclerotinia cinerea (Bon.) Wor. It is this ordinary brown-rot fungus that causes the severe attacks of fruit rot of stone fruits in Oregon. It often causes blossom blight as well. There is, in addition, a closely related fungus common on the Pacific Coast which often causes very severe attacks of blossom and spur blight and twig cankers on all kinds of stone fruits and on certain varieties of pears, but which causes little rotting of fruit. This fungus was investigated several years ago By G.B. Posey at the Oregon Experiment Station. His findings are reported in an unpublished thesis. The complete life-history of this parasite is not yet known. In tests it failed to produce the apothecial (spore-cup) stage under conditions that gave abundant development of this stage with the ordinary brown-rot fungus. It produces spores in chains very closely resembling those of ordinary brown-rot, but it differs from the latter in the appearance and manner of spore production on the host plant. In artificial cultures it has also proved to be very different from the common brown-rot fungus or other described species. We have, therefore, considered it a new species and have assigned to it the name Monilia oregonensis Barss and Posey. The disease caused by it will be referred to as Monilia blight to distinguish it from the true brown-rot disease.

The moist and cool weather of early spring is often very favorable to the infection of blossoms by the spores of the Monilia blight fungus and also to some extent by the spores of the ordinary brown-rot fungus. The method of attack and the effects are similar with both fungi except that the Monilia blight appears to work more rapidly and progress farther than the brown-rot fungus. Petals or calyx lobes and likewise the stigmas of the flower are very subject to infection. The first sign is a small brown spot on the petal, calyx lobe, or tip of the pistil.

his discoloration spreads rapidly if the weather continues moist. The disease ventually kills the parts attacked and begins to invade the base of the flower, hich then turns dark. After the flower has been killed the fungus often works own the flower stalk to the spur on which it was borne. The spur is frequently illed with all its blossoms and leaves..... and the disease may even extend to he twig, causing a small canker..... If the twig is small the canker may girdle t, resulting in the death of the parts beyond. If the weather continues moist for everal days after infection has taken place, spores often appear in delicate gray-ish tufts which give the dead flower parts a velvety appearance. If the atmosphere is too dry, however, the Monilia spores may not appear till the following winter and spring. As a rule, shortly after the blossoming period the advance of the blight is checked so that the cankers under average Oregon conditions rarely reach large extent. The black blighted blossoms and leaves shrivel, and remain hanging to the dead spurs throughout the following winter."- Barss, H. P. Brown-rot and Related Diseases of Stone Fruits in Oregon. O. A. C. Exp. Sta. Circular 53. December, 1923. pp. 4-7.

Control

General control measures are discussed by Hesler and Whetzel (2) as follows:

"In applying control measures it should be borne in mind that:

(1) All parts of the peach above ground are liable to attack;
(2) The disease is most serious in warmer regions and in warm, wet seasons;
(3) Total destruction of the crop may come suddenly and unexpectedly;
(4) The fruits and blossoms are affected seriously, which fact is of vital importance when measured in dollars;
(5) The fungus overwinters in the mummies and in the cankers, and the mummies are found both on the ground and hanging to the tree;
(6) The fungus enters fruits chiefly through wounds, and the curculio is a serious offender in making such wounds.

Therefore, remove all cankers from the larger limbs, and prune out affected limbs of smaller size. The methods followed are described under another heading. In pruning, knock off the hanging mummies. Bury the fallen mummies by plowing to a depth of several inches; this should be done before the blossoms open. The regular pruning operations assist to no little degree in the control of the brown-rot. The removal of limbs admits sunlight and air, thus allowing the susceptible parts to dry off more quickly after rains."- Hesler, Lex R. and Herbert Hice Whetzel. Manual of Fruit Diseases. pages 275-276.

Spraying.

For peaches Hesler and Whetzel (2) recommend self-boiled lime-sulfur, 8-8-50 applied: (1) about the time blossoms fall; (2) two or three weeks later or about one month after the petals drop; (3) about one month before the fruit ripens.

Fisher and Brooks (22) carried out experiments for the control of brown-rot prunes and cherries in the Pacific- Northwest. Their recommendations are cluded in the following table:

"Spray schedule for control of brown-rot on prunes and sweet cherries in western Washington and Oregon.

pplication and time	For prunes	For sweet cherries
rst brown-rot spray (bud pray). Apply when blossom uds are white and before ull bloom.	Bordeaux mixture,4-4-50: Lime -sulphur, 1 to 50.: Self-boiled lime-sul- phur, 8-8-50. Sulphur dust.	Bordeaux mixture, 4-4-50. Lime-sulphur, 1 to 50. Sulphur dust.
econd brown-rot spray calyx spray). Apply as oon as most of the petals ave fallen	Bordeaux mixture,4-4-50: Lime-sulphur, 1 to 50. Self-boiled lime-sulphur: 8-8-50. Sulphur dust.	Bordeaux mixture, 4-4-50. Lime-sulphur, 1 to 50. Sulphur dust.
ird brown-rot spray. pply as soon as husks are hed.	Self-boiled lime-sul- phur 8-8-50. Bordeaux mixture, 4-4-50. Sulphur dust.	Lime-sulphur, 1 to 50. Sulphur dust.
urth brown-rot spray. pply two to three weeks efore fruit is ripe.	Self-boiled lime-sul- phur, 8-8-50. Bordeaux mixture,4-4-50: Sulphur dust.	Lime-sulphur, 1 to 50. Sulphur dust."

Review questions.

Describe all symptoms of brown rot on the various parts of plant attacked nd on the various kinds of fruits. Describe complete life history of the brown ot fungus, especially as to perpetuation, dissemination and infection. Discuss ontrol measures in relation to the life history cycle. Compare life history and ontrol measures of Brown Rot with those of peach leaf curl.

References.

1) Duggar, B. M. Fungous Diseases of Plants, p. 187.

2) Hesler & Whetzel. Manual of Fruit Diseases, pages 140, 143, 153, 170, 270, 354.

3) Brooks, Charles and Fisher, D. F. Brown rot of prunes and cherries in the Pacific Northwest. U. S. D. A. Bul. 368. March 6, 1916.

4) Jehle, R. A. The brown rot canker of the peach. Phytopathology 3: 105. 1913.

5) Jehle, R. A. Peach cankers and their treatment. Cornell Exp. Sta. Cir. 26-53. 1914.

6) Scott, W. M. & Quaintance, A. L. Spraying peaches for the control of brown rot, etc. U. S. D. A. Farmer's Bul. 440. 1911.

7) Jackson, H. S. Brown rot of stone fruits. Ore. Exp. Sta. Crop Pest Report 1911-12. p. 248.

8) Stewart, F. C. Notes on N. Y. plant diseases, I. Cherry. Brown rot. N. Y. (Geneva) Exp. Sta. Bul. 328: 338. 1910.

9) Clinton, G. P. Apple rots in Illinois. Brown Rot. Ill. Exp. Sta. Bul. 69: 190. 1902.

10) Heald, F. D. The black rot of apples. Neb. Exp. Sta. Report 19: 82. 1906.

11) Norton, J. B. S. Sclerotinia fructigena. Sci. 16: 34. 1902.

12) _____ Sclerotinia fructigena. Trans. Acad. Sci. St. Louis 12: 91-97. pl. 18-21. 1902.

13) Norton, J. B. S., W. N. Ezekial and R. A. Jehle. Fruit-rotting Sclerotinias. I. Apothecia of the Brown-rot fungus. Maryland Exp. Sta. Bul. 256. 1923.

14) Valleau, W. D. Varietal resistance of plums to brown-rot. Journ. Agric. Res. 5: 365-396. 1915.

15) Pollock, J. B. The longevity in the soil of the Sclerotinia causing the brown-rot of stone fruits. Mich. Acad. Sci. Rpt. 20: 279-280. 1918.

16) Wormald, H. On the occurrence in Britain of the ascigerous stage of "brown-rot" fungus. Ann. Bot. 35: 125-135. 1921.

17) Roberts, J. W. The age of brown-rot mummies and the production of apothecia. Phytopath 11: 176-177. 1921.

18) Cunningham, G. H. The significance of apothecia in the control of brown-rot of stone-fruits. New Zealand Journ. Agric. 25: 225: 230. 1922.

19) _____ Plant disease in Maryland in 1902. Md. State Hort. Soc. Ann. Rpt. 5 (1902): 99. 1903.

20) _____ Plant Pathology. Md. State Hort. Soc. Ann. Rpt. 6 (1903): XLIX - L. 1919.

(1) Ezekiel, W. N. Some factors affecting the production of apothecia of Sclerotinia cinerea. Phytopath. 11: 495-499. 1921.

(2) Fisher, D. F. and Charles Brooks. Control of Brown-rot of prunes and cherries in the Pacific northwest. U. S. D. A. Farmers' Bull. 1410. 1924.

(3) Barss, H. P. Brown-rot and related diseases of stone fruits in Oregon. Ore. Exp. Sta. Cir. 53. 1923.

STEM ROT OF CLOVER AND ALFALFA

Caused by Sclerotinia trifoliorum Eriks.

This is a serious disease of clover, especially in Europe. In America, also t is serious at times. The mycelium invades the stem causing it to decay near he surface of the ground. The mycelium also spreads over the surface of the in- ected parts as a whitish moldy growth. Soon sclerotia appear and these later ive rise to the cup or apothecial stage which produces ascospores. No conidia re known.

This disease may attack alfalfa and other legumes as well as clover.

1. Laboratory study.

a. Symptoms.- Examine diseased stems and note effect. Note mycelium and clerotia if present. Draw.

b. Life history.- Examine cups (apothecia) growing from the small black clerotia which have lain on the ground for some time. Draw. Compare with cups f brown rot previously studied. Mount bits of hymenium from cup and draw asci.

Discussion.

The historical account immediately following is contributed by Gilbert and Iyer (4).

"Historical.- The 'stem rot' of clovers and alfalfa (previously referred to lso as 'clover sickness') has been known in Europe and England for more than ifty years and has there been the subject of considerable investigation. Accord- ng to Erikson, a German authority, the first recorded occurrence of the disease as in Germany in 1857, and the first case in which clover sickness was attributed o the fungus under consideration as the cause was reported by Hermann Hoffman in 863. In 1872 a paper containing a very full description of the disease and a ife history of the causal organism, together with suggestions as to treatment, as published by Emil Rehm of Gottingen, Germany. This paper pointed out that the isease was a very serious one in certain sections of the country, and that it was especially difficult to control on account of the settled custom of the farmers here to grow much alfalfa (lucerne) and clover, and on account of the practice of

ping fields in clover for a period of several years. This practice encouraged disease to become better established and to spread. The only recommendation the author was the rotation of crops so as to remove from the diseased fields crops which served as hosts for the fungus. After several years during which land had been in other forage crops, clover and alfalfa might again be grown h safety.

"In England investigations of 'clover sickness' by Lawes and Gilbert date k to 1849 and were continued through nearly thirty years, but it was not until 08, according to Gussow, that the cause of the disease was there definitely ribed to a fungus. In addition to its occurrence in England and Germany, this ease has also been reported from Sweden, from Denmark, and from Canada. In ada, Gussow reports only one case of the disease but is very much concerned as its spread.

"Although in Europe the disease has been prevalent for many years, in this ntry it is of more recent occurrence, and has not as yet become so great a ace as it is abroad. Chester of Delaware, in 1890, seems to have been the st to report upon the disease in this country."- Gilbert, A. H. and D. S. Meyer. em Rot of Clovers and Alfalfa as a cause of "Clover Sickness". Kentucky Agricultural Experiment Station Circular 8. pp. 48-49. September, 1915.

Further items on stem rot are contributed by Wolf and Cromwell (5) as llows:

"Distribution of the Disease.- As indicated above, the disease is known to cur abroad in Germany, Denmark, Sweden, and England, and in America within laware and Kentucky. It has furthermore been recorded from Canada, New York, w Jersey, Virginia, Indiana, Oregon, and North and South Carolina. Its prevance in these states indicates that it very probably occurs in other of the ates where clover is grown.....

"Host Plants.- The disease has been shown by European writers to attack red over, white clover, crimson clover, alsike clover, sainfoin and alfalfa. To is list Gilbert and Myer have added spotted spurge, Euphorbia masculata. Frank served a sclerotial disease of peanuts of whose identity he could not be certain nce he did not have the apothecial stage, but he may have been dealing with the me organism.....

"Description of Clover Stem Rot.- Stem rot on crimson clover can first be ticed in the fall when the plants are still small. It continues to spread, wever, when weather conditions permit, throughout the winter months and is structive until the time when unaffected plants are mature. The first evidence the presence of the disease is manifested by a wilting of the stem and leaves. ese portions then turn yellowish, rather quickly succumb and become brown. oser examination discloses the fact that the stems near the base of the stool at the ground level are involved in decay, which begins as a slight discoloration and proceeds until the stems have rotted off. At this time a dense, white, ld-like growth is prominently present at the base of affected stems. In the esence of a suitable supply of moisture this fungous growth becomes more profuse d within a few days, compact masses of hyphae will have formed on the surface the decaying stems. These masses soon become black in color and of a cartilagious consistency and are the sclerotial stage of the causal organism.

"These black sclerotia are sometimes as large as a pea and their presence
n be used as an aid in a field diagnosis of the disease. With the maturing of
ese sclerotia and the simultaneous disintegration of the stems, the sclerotia
main scattered over the soil where the plant has been. Meanwhile, the roots
so are involved in decay and the sclerotia which are formed as the tissues
come decomposed, remain in the soil. Since the disease spreads outward from a
nter of infection, plants in all stages of the disease may be found in any one
ot. When the diseased areas are large the centers may be entirely bare or there
y remain the debris of the badly decayed stems. The stand of clover, in case
ants die in localized areas, is thus rendered not uniform or when the centers
infection are numerous and extremely favorable conditions prevail, the stand
y be rather uniformly destroyed.....

"Life History of the Causal Organism.- The life cycle of the clover stem rot
ngus is relatively simple since it possesses only one functional type of spore.
lives for the greater part of the year as a saprophyte and can maintain itself
the soil upon decaying plant tissues from one year until the next. On the basis
difference in appearance and function, the fungus may be regarded as possessing
ree stages. Under normal conditions the first or vegetative stage is present
ring late fall, winter, and early spring, within the clover plants causing their
ath. Invasion of the tissues begins at the collar, near the ground level, and
tends upward and downward from this point of attack. During the same period the
ngous continues its vegetative growth as a saprophyte in plants which it has
lled. As the tissues are disintegrated the fungus appears at the surface of the
caying parts and becomes massed together in compact black sclerotia, the second
age. These sclerotia then remain dormant on the surface of the soil or within
e soil in the spots left bare by the destruction of the clover. Since these
lerotia require only two or three weeks to mature they are being formed practi-
lly throughout the entire growing period of the clover crop. These sclerotia,
ich contain a reserve of nutriment, remain dormant and serve to carry the fungus
er the summer season, or until opportune conditions for their growth again pre-
il. During the fall and early winter these sclerotia germinate by the formation
slender, yellowish-brown stalks. The distal ends of these stalks expand into
e disc-shaped fruit bodies, which with the stalks constitute the apothecia, the
ird stage. From one to a half dozen apothecia are developed from each sclerotium,
pending upon the size of the sclerotium. The upper or inner surface of these
iscs is lined with a layer of closely aggregated, elongated sacs, or asci, inter-
persed with the paraphyses. Rehm has estimated that there are approximately
,000 sacs in one square millimeter of surface, each of which bears at maturity
ight ascospores. These ascospores are forcibly discharged, often in such numbers
s to appear like small clouds of dust. They may then be carried by the wind to
moist surface and there germinate at once by the formation of a hypha. This
ypha becomes, as growth proceeds, the vegetative mycelium, thus completing the
ycle of development.

"Whether these ascospores directly infect clover is not known, aside from the
xperimental work of Rehm and Coleman. The former suspended mature fruit bodies
er healthy plants and within 6 to 8 days the mycelium was present within the
eaf blades, the plants subsequently collapsed, and sclerotia were formed in due
ime. He did not determine the mode of entrance of the germ tubes, but regarded
t as probable that they entered through the stomates. The latter reported that
oung clover plants only can be readily infected by ascospores, although under

vorable conditions older plants were in a few instances attacked. His investi-
tions furthermore led him to assert that infection does not take place through
e stomates, but by penetration of the epidermal cell walls..... Because of
e fact that infection begins on the stems at or near the ground level in the
se of stem rot of clover, it is believed that the vegetative mycelium is respon-
ble for the disease and that little disease results naturally by direct in-
ction with ascospores.....

"Means of Spread of Stem Rot.- It seems highly probable that stem rot of
over was introduced into America from Europe, but the method of its introduction,
gether with the means for its spread into new localities each year, have remained
re or less problematical. Eriksson states that the fungus undoubtedly over-
nters as hyphae adhering to the seeds and not as sclerotia or spores and that
e disease is spread by this means.

"Coleman suggests that sclerotia mixed with the seed are a probable means of
stribution, although spores adhering to the seeds are also a possible means.
servations on this point, first made during the fall of 1914, by Professor H. R.
lton, then Plant Pathologist for the North Carolina Experiment Station, show
at sclerotia are present in commercial crimson clover seed. He states: 'I re-
ived from county agents some six or eight samples of seed. Two of these, I
stinctly remember, showed the contamination with sclerotia.' If seed were
rvested from affected fields, sclerotia would almost certainly be mixed with
ed, and would remain with them even though the seed were very carefully cleaned.
ed contaminated with sclerotia are, therefore, believed to account for the in-
oduction and presence of stem rot of clover.

"Numerous possibilities might be suggested to account for the local distri-
tion of the disease. The fungus is known to spread through the soil along the
rgin of the diseased areas. Further, the sclerotia which normally remain
rmant for a time, even though favorable conditions for their germination may
tain and which may retain vitality for at least two and one-half years under
boratory conditions and one and one-half years in the soil may aid in its spread.
ey may be distributed by the implements used in cultivation, by the use of soil
inoculating with the nodule forming organism, by rains, or by being harvested
th the hay crop. If this hay is fed to stock the possibility exists of the
turn of these sclerotia to the fields through the manure.

"The violent discharge of ascospores from the mature apothecia and the car-
iage of these spores by the air currents may also be taken into account in the
cal spread for short distances of the stem rot fungus.

"Method of Control..... Precaution should be exercised both in the saving
f seed from affected crimson clover fields and the planting of such seed because
f its contamination with sclerotia. To guard against the introduction of stem
ot into new fields or new localities, seed should be sampled and sent to a seed
esting laboratory in advance of purchase. The introduction of the disease through
anure from stock fed on hay from affected fields is also to be guarded against.

"It is known that not all host species are equally subject to attack by S.
rifoliorum, but whether or not any one of these species possesses marked varietal
ifferences in susceptibility is not known. No experimental work appears to have

n done on selection for disease resistance and the only observation that has
ıe to our attention that such differences exist is by Nilsson-Ehle who records
.t Swedish red clover of the variety serotinum is more resistant than exogenous
·ains.

"In fields badly infested no measures can be relied upon to be effective
ıer than the adoption of a proper rotation system in which clovers are not grown
· a period of three or four years. Definite data are wanting on the length of
ıe which the stem rot organism can live saprophytically in the soil, but it is
;ical to assume that it would no longer be present or the infective material
ıld at least be greatly reduced in amount within a lapse of three or four years.
rpeas and soybeans which are not subject to attack by Sclerotinia trifoliorum
y well be used as a leguminous crop in such a system of rotation and winter oats
l rye can be employed as winter cover crops."- Wolf, F. A. and R. O. Cromwell.
›ver Stem Rot. North Carolina Agricultural Experiment Station Bulletin 16.
. 6-15. June, 1919.

Review.

Compare in all details the stem rot of clover with the brown rot of stone
uits. How are the diseases alike? How different?

References.

) Duggar, B. M. Fungous diseases of plants (Text) p. 201.

) Stevens & Hall. Diseases of Economic Plants (Text). p. 386.

) Jackson, H. S. Wilt or Stem Rot of Clover. Ore. Exp. Sta. Crop Pest Report, 1911-12. Page 307.

) Gilbert, A. H. & Myer, D. S. Stem rot of clovers and alfalfa. Ken. Exp. Sta. Cir. 8, September, 1915.

) Wolf, F. A. & Cromwell, R. O. Clover Stem Rot. N. C. Exp. Sta. Technical Bul. 16. June, 1919.

LEAF SPOT OF CLOVER AND ALFALFA

Caused by Pseudopeziza trifolii & P. medicaginis

1. Examine a diseased leaf first with the naked eye or dissecting microscope,
en place it under the binocular microscope. Note the small disc-like fruiting
dies (apothecia) protruding through the epidermis in the center of the dark
ots. Make habit-sketch of whole leaf natural size, then of one apothecium en-
rged as seen under the binocular microscope.

2. Examine a prepared amount of a section through an apothecium. Note layer acs, (hymenium). Draw.

3. Write notes.

References.

Duggar, B. M. Fungous Diseases of Plants. p. 203.

Combs. Robert. The Alfalfa leaf spot disease. Iowa Exp. Sta. Bul. 36: 858. 1897.

Jackson, H. S. Leaf Spot of alfalfa and clover. Ore. Exp. Sta. Crop Pest Report. 1911-12. p. 305.

Jones, F. R. The leaf-spot diseases of alfalfa and red clover. U. S. D. A. Bul. 759. June 19, 1919.

Review.

Compare the leaf spot disease of clover and alfalfa with the stem rot disease the same plants.

NORTHWESTERN ANTHRACNOSE OF APPLE

Caused by Neofabraea malicorticis (Cordley) Jackson

Anthracnose is by far the most prevalent canker disease of apple trees in e Pacific Northwest. Especially is this true of the Willamette Valley where glected orchards are sometimes practically destroyed by this disease.

1. Laboratory Study.

a. Symptoms.- Examine specimens of cankered branches and of rotted fruit, d note all symptoms and signs of the disease on both. Note the pustules where e fruiting stage of the fungus breaks through the epidermis. Examine these stules with a hand lens, a dissecting microscope or a binocular microscope. ke sketches of both diseased branches and fruits. Make drawing of one pustule cervulus) greatly magnified. Describe all symptoms in notes.

b. Life History and Morphology of Causal Organisms.

(1) Conidial stage.- Examine, under the microscope, sections cut rough the pustules of a one-year-old canker, or of a rotted fruit. Find the ore-bearing stalks (conidiophores) and the spores (conidia) which they bear. ke a drawing of the section to show relation of fungus to host tissue. Draw a nidiophore and conidia enlarged.

(2) Perfect Stage.- On an old two-year-old canker find the small eshy, disc-like fruiting bodies (apothecia). These fruiting bodies of the perfect age bear spores in sacs (asci) instead of on conidiophores as is the case in the nidial stage. Each apothecium has its upper surface covered with a layer of ci just as were found in the cups of the brown rot fungus.

Examine sections cut through bark and apothecia. Draw to show relation of ngus to host. Show layer of asci (hymenial-layer) on upper surface of apothecium

2. Field Study.

If circumstances permit, visit an apple orchard where anthracnose is resent. Observe any of the following features which are available and take full otes.

a. Early stages in development of cankers. Note small, roundish or oval, ark-colored spots on the bark. No pustules are yet visible but bark is perfectly mooth. This stage can be observed through the winter and early spring months.

b. One-year-old cankers. These show conidial pustules breaking through uter bark. Note sunken appearance due to drying out of dead bark and swellings f callus around margin of cankers. Bark is more or less cracked loose around argin of canker.

c. Cankers two or more years old. In some of these the dead bark may till be clinging. Look for pustules larger than on the one-year-old cankers. ind the waxy or fleshy discs in these pustules. These are the fruiting bodies f the perfect stage.

d. Bring material of both the conidial and perfect stage into the labor- tory and examine to see if mature spores are present.

3. Notes.- Write up in detail all information obtained from laboratory and ield study. Add any points of interest obtained from reading the following eferences.

Discussion.

The chief facts known about apple tree anthracnose are set forth in the ollowing quotations from Jackson's (2) article in the Ore. Exp. Sta. Crop Pest Report for 1911-12.

"The apple tree anthracnose is, with the possible exception of the apple scab, he most serious fungus disease with which apple growers in the northwest have to deal. In general, the disease is characterized by the formation of dark colored sunken cankers in the bark of the younger branches. These are most abundant on branches under two or three inches in diameter, but are not uncommonly found on branches of larger size.

"When they occur on older branches having a thick bark, the cankers may not lways penetrate entirely to the wood. Usually, however, the bark in the mature ankers is found to be entirely dead, the cambium destroyed and the sap wood dis- olored to a limited extent.

Development of the Canker

"By close observation one may find the young cankers beginning to develop ost commonly early in November. The time when the cankers start doubtless varies reatly with the season. It is possible that in seasons of early fall rain they ay begin to appear much earlier.

"They begin their development as small reddish-brown spots in the bark. These hen first observed are circular and about one-half inch in diameter and are not unken. If one cuts into the bark beneath one of these spots the tissues are found o be discolored and to present a water soaked appearance. The discolored area is ound soon to extend to the cambium, where it spreads out and may in some stages f the development of the cankers be more extensive in that region than the dis- olored area on the surface would indicate. The spots develop very slowly, if at ll, during the winter months, but spread quite rapidly during March and April. s they enlarge they gradually become elliptical in outline, the surface becomes lat, then slowly sinks.

"Sometimes the bark of developing cankers shows bands of slightly varying olor, giving the appearance of concentric zones. This is doubtless due to alter- nating period of rapid and slow growth induced by variations in the weather con- ditions. In early stages of rapidly growing cankers an irregular crack may appear n the bark and drops of fermenting sap may exude.

"In the early spring, when the warm weather induces renewed activity in the hysiological processes of the tree, the cankers develop rapidly. The bark soon dries and sinks and this condition together with the normal growth in the surround- ing healthy bark produces a tension which results in the formation of a crack in the bark at the edge of the canker. The spread of the fungus ceases as soon as the cambium becomes active in the spring.

"After the cankers reach the full size, which occurs in early May, further activity is confined to changes which take place in the dead bark within the canker. If a mature canker is examined in midsummer, little elevations in the bark are easily observed. They are at first more or less conical in shape and finally burst the outer layer of the bark exposing a cream colored mass of fungous tissue. These are the fruiting structures of the causal fungus and the spores are produced in them in great abundance.

"In late summer or early fall the mature cankers are found in abundance and may be from one-half inch in diameter up to eight to 10 inches long by three to four broad. Frequently large cankered areas may be formed by several smaller ones becoming confluent. Mature cankers have a definite limiting crack separating them from the healthy tissue, there is usually a definite ridge surrounding the canker caused by the slight formation of callus tissue at the edge under the diseased bark. The bark is sunken, dry and dead, and darker in color than healthy bark.

nickly scattered over the surface one finds the little cracks described above nich are formed when the fungus bursts forth. These cracks are transverse or riangular, seldom if ever, vertical..... In old cankers the fungus tissue xposed by the cracks turns black and the bark gradually becomes loose at the dges and drops out, leaving ugly wounds. Bark may cling in the cankers, however, or at least three winters. The wounds, if not too large, slowly heal over by ne formation of callus.

Injury Caused by the Disease.

"On account of the nature of the disease it is difficult to estimate the mount of the injury resulting from its ravages. Under ordinary conditions few o many cankers may be found on the branches of the trees in infected orchards. ometimes twigs are girdled by the formation of a small canker, which extends round the stem..... Not infrequently larger branches are girdled where several ankers grow together. Sometimes trunks of young trees are girdled in this way.

"It is evident that the disease in any degree of severity interferes with he normal function of the bark and so hinders the proper distribution of elaborted food in the tree. Branches are weakened by the presence of the cankers and requently break when heavily set with fruit. The wooly aphis not uncommonly orks under the bark at the edge of cankers and further saps the vitality of the ree and interferes with the normal healing over of cankers. Large cankers heal lowly and the wood may be exposed for considerable periods, thus affording opporunity for the entrance of fungi which cause heart rot.

Distribution.

"As a serious orchard disease the apple tree anthracnose is peculiar to the acific Northwest. It is known to occur in British Columbia, Washington, Oregon nd Idaho. It has not been recorded in California to our knowledge, though it is ot improbable that it occurs in the norther counties, since it is common in ackson and Josephine counties in Oregon. It is most serious in those sections aving considerable rainfall west of the Cascade mountains. It is rarely a erious disease in the fruit sections of Eastern Oregon.

Life History Studies.

Results of Previous Investigators.

"The apple tree anthracnose has probable occurred in the northwest for many ears. It began to attract attention as an orchard trouble of some consequence uring the period from 1891 to 1893.....

"In 1899 following insistent demands upon the Oregon Agricultural Experiment tation for information regarding this disease, Cordley, Entomologist to the xperiment Station, undertook an investigation of this disease..... As a result of this investigation Cordley discovered the true cause of the disease which he

found to be a previously undescribed fungus. He isolated this fungus, studied it in pure culture and produced typical cankers on apple branches in the laboratory. Under date of January, 1900, he published a short, more or less popular, bulletin (3) giving the results of his investigation and experiments and recommending a method of treatment. In this publication he referred to the causal fungus as Gloeosporium malicorticis....

"In 1904 Lawrence, at that time assistant botanist at the Western Washington Experiment Station located at Puyallup, published a detailed account of the apple tree anthracnose under the name of 'black spot canker'.

"In this bulletin Lawrence confirms the work of Cordley in regard to the life history of the fungus and its pathogenicity and describes in detail the development of the canker and records many interesting and important biological phases of the disease. The interesting information that the fungus also causes a rot of stored fruit is given and proven by isolation and inoculation experiments. He also shows by experiment that the fungus is disseminated most commonly by the wind and that it is able to penetrate the bark of branches in the absence of any injury, most commonly through the lenticels. Inoculation experiments on hosts other than the apple are recorded.....

Previously Unpublished Investigations.

"..... Early in the work a search was made for a possible ascogenous stage in the life history of the fungus. In November, 1909, while studying the characters of 'two year old' cankers, that is, cankers resulting from infection in the fall of 1907, the apothecia of a discomycete belonging to the family Mollisiaceae were found occupying the position of the acervulus of the previous season (1908). Careful search revealed the fact that the apothecia of this fungus were nearly always found in the dead bark of cankers one year after the development of the conidial stage. The writer has had this fungus under observation now for three seasons and has never failed to find it in the bark of cankers two years after infection in any orchard in which he has searched at the proper season.....

"From an examination of the characters of the genera of the Mollisiaceae and related families, the writer has failed to find any genus having those characters. Therefore, the following new genus is proposed for what seems without doubt to be the ascogenous form of Gloeosporium malicorticis.....

(At this point Jackson gives a technical description of the perfect stage of the fungus, which he has discovered, and proposes the name, Neofabraea malicorticis (Cordley) n. comb. He then continues the discussion as follows:)

"The apothecia stand out prominently during the rainy weather and tend to disappear during the dry weather, swelling up again with the next rain. On this account the normal production of spores is frequently interfered with and there is a tendency for the spores to become septate and germinate within the ascus, sending threads to the surface of the ascogenous layer and there developing secondary conidia. When the conditions, however, are favorable for any length of time, the asci containing the matured spores elongate and extend for about one-half their length above the surface of the apothecium. The ascus swells and

ores collect in the apex and finally are ejected by the ascus bursting and attering the spores forcibly. This process was observed repeatedly under the croscope.....

Disease on Fruit.

"The apple tree anthracnose is not uncommon as a rot of the fruit. This may developed in the orchard, especially if the fruit is allowed to hang late on ne trees, or may be developed as a storage rot on fruit which is perfectly sound nen stored.....

"Cate also found the disease common upon apple fruit and produced the rot by noculation. The writer has confirmed the more important results of these inves-igators and has also produced a rot on the fruit by inoculation with cultures otained by germination of ascospores. In the season of 1911 this disease devel-oed abundantly and seriously as a rot on stored fruit both under ordinary storage onditions and in cold storage. The writer has seen boxes of Spitzenburg apples ept in storage until May in which 90% of the fruit was affected with this disease. he disease seems to be more abundant on the fruit, as would be expected, in easons of early fall rain or when on account of weather conditions picking is elayed.....

Summary of Results.

"1. A disease known throughout the Northwest as Apple Tree Anthracnose or lack Spot Canker is caused by a fungus which, in the conidial stage, is a para-ite causing cankers on the branches of apple and pear trees.

"2. The cankers caused by this fungus cause the death of the tissue of the ark in which it develops and of the cambium, and spreads to some extent to the apwood beneath.

"3. Infection occurs in the fall and early winter and the cankers develop lowly during the early part of the dormant season and spread rapidly in the arly spring, reaching their full size when the cambium resumes activity in the pring.

"4. The fungus matures slowly during the summer in the dead bark of the ankers thus formed. Spores are produced in acervuli characteristic of the enera Gloeosporium or Myxosporium of the Fungi Imperfecti.

"5. This conidial form has been named by Cordley, Gloeosporium malicorticis.

"6. The fungus continues to develop in the dead bark of old cankers as a aprophyte and produces in the late summer or fall, one year after the maturity f the conidial stage, an ascosporic form having the characteristics of the family ollisiaceae of the Discomycetes.

"7. The connection of this Ascomycete with the conidial stage has been proved inoculation experiments.

"8. This has been made the type of a suggested new genus called Neofabraea. proper scientific name for the fungus should now be Neofabraea molicorticis. ordley).

"9. The conidial stage has been found commonly causing a rot of the fruit the apple and the quince, both in the orchard and in storage.

"10. Both the conidial and ascosporic stages have been found in nature on the ar. Cankers have been produced on this host by inoculation with the conidial age of the fungus isolated from the apple.

"11. Cankers have also been produced by inoculation with the fungus isolated om the apple, on peach, prune and cherry branches, but no spores have been ob- rved to be matured in these cankers. The occurrence of such cankers in nature s not been proved.

"12. No evidence has been secured that new cankers are formed by the spread the fungus in the tissues from old cankers. The fungus has, however, been olated from the heart wood beneath cankers on branches of the apple, two years ter the maturation of the conidial stage. The extent and importance of the ngus as a heart rot has, however, not been investigated thoroughly."- Jackson, S. Oregon Agricultural College Biennial Crop Pest and Horticultural Report. 11-12. p. 196.

Control.

The following discussion of control measures is quoted from Barss (9).

"Because the rainy fall and winter season is the infection period for this isease the control methods used must be such as will insure protection for the ee and fruit during this period. There are two ways in which protection may be ought about: first, by removing all sources of infection and, second, by cover- ng all susceptible parts of the tree and coating the fruit with a spray which is oisonous to the fungus.

"Surgery.- Cutting out the cankered areas or removing seriously injured anches is practicable only to a limited extent. Where very young trees can be arefully inspected considerable benefit may be derived by watching for and cutting ff all cankered areas and painting the wounds with Bordeaux paste. When old trees re very severely attacked it may be worth while to resort to surgery but complete emoval of all cankers is impossible and spraying must be resorted to anyway.

"Spraying.- Remarkable successful results are obtainable from spraying. ractically complete prevention of tree infection will occur where one thorough pplication is given in late summer before the moist period of the fall starts in. ordeaux 4-4-50 applied in the August codling moth spray has resulted in almost erfect protection in many orchards threatened with complete destruction from the isease. The use of this application so late in the summer makes necessary the

ing of the fruit. When the autumn is very wet there is also some danger that ayed fruit will show a tendency to develop the not uncommon red speckling or ticel injury more promptly than on unsprayed fruit but the evidence at hand licates that this difficulty and the cost of spray and wiping are more than set by the protection against rot which the presence of the spray on the fruit ords. Every twig, branch and limb as well as the trunk should be completely ted for perfect results.

"In the past in orchards where anthracnose had not yet got an excessive hold, asonable successful control has been secured by putting on Bordeaux 6-6-50 just soon as the fruit was picked. Labor scarcity, delayed harvesting and bad ather have interfered so much of late with this after harvest spray that many chards have got into very bad shape and growers will now be compelled to put on protective spray before the fall sets in or run the risk of almost complete ruin trees and crop.

July Spray.- It is believed that equally effective anthracnose canker control n be obtained by using Bordeaux 4-4-50 with the July lead arsenate spray instead the August spray. There will result less objectionable deposit on the fruit harvest. The Hood River Branch Experiment Station is now testing the use of rdeaux and oil in the delayed dormant application just as the winter buds are ening in the spring as the one annual spray for anthracnose and first spring ray for scab, leaf-roller and other troubles. These tests have not reached the int where definite recommendations can yet be made but there are interesting ssibilities which we hope may be realized in the final report."- Barss, H. P. ple tree anthracnose canker and rot. Ore. Agri. Col. Extension Service Cir. 0. May, 1922.

Review.

Describe the symptoms of anthracnose on both branches and fruit. Discuss mpletely the life history cycle. How, when, and where do new infections take lace? Discuss control measures as related to life history. Can complete control e obtained? To what part of the country is this disease confined?

References.

1) Hesler and Whetzel. Manual of Fruit Diseases, p. 81.

2) Jackson, H. S. Apple Tree Anthracnose. Ore. Exp. Sta. Crop Pest Report 1911-12, p. 178.

3) Cordley, A. B. Apple Tree Anthracnose. Ore. Exp. Sta. Bul. 60. January 1900.

4) Jackson, H. S. Apple tree anthracnose. Ore. Exp. Sta. Cir. Bul. 17. Sept. 1911.

5) Cordley, A. B. Some observations of apple tree anthracnose. Bot. Gaz. 30: 48. July, 1900.

Cordley, A. B. Apple Tree Anthracnose. Better Fruit. Vol. I, No. 5. November, 1906.

Lawrence, W. H. Black Spot Canker. Wash. Exp. Sta. Bul. 66, 1904.

Cate, C. C. Apple Tree Anthracnose. Oregon Countryman, Vol. I, No. 2, October, 1908. Also Vol. I, No. 3, November, 1908.

Barss, H. P. Apple tree anthracnose canker and rot. Ore. Agric. Coll. Extension Circular 160. May, 1922.

THE POWDERY MILDEWS

Family Erysiphaceae

This family of the Ascomycetes is responsible for a very troublesome disease a great many plants. Some of the more common plants that are subject to the acks of powdery mildews are: apple, peach, rose, gooseberry, wheat and other ins, peas, grape, hop and many uncultivated trees and plants of field and est.

The following description, taken from Duggar's Fungous Diseases of Plants, lies to the powdery mildew family as a whole.

"Morphological.- With only one or two exceptions (notably Sphaerotheca Mors-e) the superficial mycelium of these plants consists of colorless hyphae, con-erably septate, each cell being ordinarily uninucleate. In all species except , so far as is known, the haustoria penetrate the epidermal cells in the form short, swollen branches. However, in one common mildew of shrubs and trees yllactinia corylea) hyphal branches grow through the stomata and into the inter-llular spaces. These branches may in turn develop haustoria, which enter the ls in contact with this intercellular hypha. As a rule conidial production in forms begins whenever a considerable mycelium has been developed. These nidia consist, quite generally, of a single chain of cylindrical or more or less rrel shaped unicellular portions produced in basipetal order on short, erect nidiophores, developed directly from a hyphal cell. The conidia are capable of nediate germination, and since they are produced in quantity, they frequently ve the mealy or powdery appearance to the parts affected. They serve for the oid propagation of these fungi. The conidial stage was for a long time uncon-cted with the perithecium form and was then known under the form-generic name dium. The minute characteristics of the oidial stages have not been sufficiently ndied. It is proper to use the name Oidium for any conidial form the perfect age of which is unknown or undetermined.

"Perithecia are usually developed during the middle or latter part of the owing season. They are produced directly upon the mycelium..... From the ll cells of the perithecium there are produced, either from the base or from a re or less equatorial plane, the characteristic appendages. In a few cases ly are appendages produced from the apex. At maturity there are one or more

, depending upon the genus, and each ascus contains normally from two to t spores, the shape of the ascus varying from practically spherical in the ascus forms to clavate or cylindrical where there are two or many asci. The es are one celled and colorless. As a rule the ascospores do not germinate diately, requiring a period of rest. By the following spring the perithecia very brittle and are said to break open forcibly in water, after which time ascospores readily germinate. The appendages of the perithecium are very erent in structure from the mycelium in general. The thick walls, rigidity he cells in most genera, and peculiar branching indicate that they are ialized structures, and they doubtless have an importance in relation to the ort of the perithecium or the dissemination of this body."- B. M. Duggar. ous Diseases of Plants. 1909: 218-221.

POWDERY MILDEW OF WHEAT

Caused by Erysiphe graminis D. C.

This species of mildew, or various races or strains of it, occurs on many he grains and grasses, including wheat, oats, barley and rye.

1. Laboratory Study.

a. Symptoms.- Examine leaves and stems of wheat or other grain infected mildew. Note the growth of mycelium on the surface. In most of the powdery lews the mycelium does not penetrate the host deeper than the epidermis. Is true of the "downy mildews"? Note the black specks scattered here and there he tufts of mycelium. These are the fruiting bodies (perithecia) of the fect stage. Make sketches.

b. Life history.

(1) Conidial stage.- Mount, in a drop of water on a slide, some of fungous growth from the surface of the host plant. Find the straight, un- nched conidiophores, each with a chain of conidia growing on its outer end. w.

(2) Perfect stage.- Mount some of the small, black fruiting bodies examine under the microscope. Note the appendages. Press gently on the cover ss (Be careful not to break it) until the fruiting bodies break open so as to ose the enclosed sacs. How many sacs in a perithecium? How many spores cospores) in an ascus? Draw.

c. Notes.- Write up complete description of all points learned about the dery mildew of wheat.

References

Stevens & Hall. Diseases of Economic Plants, p. 375.

Reed, G. M. Powdery mildews of Avena and Triticum. Mo. Exp. Sta. Research Bul. 23. 1916.

POWDERY MILDEW OF APPLE

Caused by Podosphaera leucotricha (E. and E.) Salmon

This pest is known all over the country and it becomes quite serious in many ions of the country if allowed to go unchecked.

1. Examine diseased leaves and twigs and take notes on appearance and effect he disease. Look for both stages in the life history of the fungus. Can you l them?

2. Write-up.

Read the following references and complete your notes on powdery mildew apple.

Discussion

That powdery mildew of the apple is of considerable importance in some sec- ns of the country is shown by the following quotations from Farmers' Bulletin 0, by Fisher (7).

"Economic Importance of Apple Powdery Mildew.

"..... The disease frequently stunts the fruit and produces a disfiguring seted appearance that greatly detracts from its market value. The chief loss, ever, results from its attacks upon the foliage, which, in severe cases, so italizes the tree that it fails to produce fruit buds and causes a crop failure the succeeding season.

"The prevalence and severity of the disease in any season vary with the matic conditions. When humid conditions prevail the spread of the disease is atly facilitated, and it often assumes, if unchecked, epidemic proportions re it has become established. It is usually more common in the spring than ring the summer, on account of the greater prevalence of rainy weather.

"The disease is most important upon the apple, but also occurs on the pear, nce, hawthorn, cherry, plum, and juneberry. The commercial varieties of apple st susceptible to the disease are the Jonathan, Yellow Bellflower, Yellow town, Esopus (Spitzenberg), Black Ben (Black Ben Davis), Grimes, and Rome auty. No variety is entirely immune, but the White Pearmain is the least sus- otible and the Winesap is also resistant.-

"Description of the Disease

"The disease is found on the foliage, twigs, blossoms, and fruit of the apple.

"Upon the affected leaves the disease is first manifested in small grayish or ite feltlike patches of fungous growth. It usually appears first upon the under

of the leaves, which soon become crinkled and curled. When very young leaves attacked they have a tendency to increase in length but not in breadth and may me somewhat folded longitudinally. The fungous patches rapidly enlarge and cover the entire leaf. In a very short time the affected areas are covered masses of powdery spores, which give the disease its name and spread it about ng the growing season. The affected foliage is rendered hard and brittle and requently killed. In any case it ceases to function normally, and a devitali- on of the tree with consequent crop reduction results, the severity of which nds upon the proportion of the leaves attacked.

"The tender twig growth may be infected independently, but is frequently cked by the fungus spreading from attached leaves. Spores are produced on twigs the same as on the leaves, and in addition another type, the ascospore, winter spore, is produced upon the twigs. These winter spores are contained in tiny globular black bodies that produce a speckled appearance in the felty gous covering of affected twigs as early as the middle of June. The winter res are no longer important in carrying the disease over winter, since the gous threads penetrate and remain in the new buds which form on affected twigs. the spring, when these infected buds open, the fungus resumes activity. The ection of twigs results in stunting or killing the parts affected, especially terminal growth, which is particularly susceptible.

"Blossom infection usually results from the overwintering of the fungus in dormant blossom bud. In this case the entire blossom cluster, with attendant ves, is attacked. The floral parts are shriveled and blighted, so that no it is produced. On account of the fact that infected buds do not open and ead mildew spores until some time after healthy buds have unfolded (usually ut the time normal blossoms are dropping their petals), there is little danger a general "blight" spreading over the blossoms during the blooming period.

"The young fruits, however, are frequently attacked shortly after the blossom- period, and infections may remain established on the apples until the skin dens in midsummer, after which the fungus generally does not persist upon the it. The fungus may become established upon the apples either by spore germina- n upon the fruit or by spreading down the stem from infected twigs. In the mer case it is usually the calyx end of the apple that is affected, but in the ter the basin alone may be involved. When very young apples are infected their wth is stunted, but a russeting of the skin always results from the presence the fungus. This russeting shows a tracery of fine lines, usually as a network, sometimes so closely woven that a solid patch appears. The expansion of the wing fruit frequently causes cracks to form in the hardened area and allows the le to become shriveled.

"Cause of the Disease

"Apple powdery mildew is caused by a fungus which grows superficially upon e leaves, tender twigs, blossoms, and fruit of the apple. The felt-like cover- g of fungous threads over these parts sends down at frequent intervals tiny uckers" to invade the cells of the host. These "suckers" extract the apple ices for the sustenance of the fungus. To propagate itself the fungus produces sses of spores in great profusion. These are wafted about by the wind. If one

ges upon tender growth where necessary moisture is present it will proceed to minate and start a new infection.

"This fungus has a very low moisture requirement for spore germination and refore has adapted itself to growth in arid climates where other fungous eases of the apple have not been able to persist. The occasional light rains the spring are sufficient to give it a start, and the prevalence of dews during growing season provides adequate moisture for it to continue to spread. With increasing use of irrigation water and the increasing amount of transpiration om vegetation grown under irrigation the humidity of the air in these orchards been increased. This, combined with the marked diurnal variation in tempera-re which characterizes such climates, brings dew nearly every night during the mmer, and consequently conditions for the spread of mildew are becoming more vorable.

Control Measures.

"Where it is necessary to spray for apple scab and where lime-sulphur or her sulphur sprays are used for this purpose, the scab treatment generally suf-ces for mildew control. Throughout the range of apple scab, therefore, mildew ldom requires special treatment. But in other localities, especially in arid gions, mildew must be treated independently.

"Powdery mildew has been under investigation and experimental work on control asures has been carried on in such districts by the Office of Fruit-Disease vestigations since 1907. The recommendations for control made in this bulletin e based upon this work. During the early part of this period of investigation e work was carried on in the Pajaro Valley, California, where peculiar local nditions make the disease a most serious menace to the apple-growing industry d where local climatic influences necessitate special treatment to effect con-ol. Later work was carried on in the Wenatchee Valley, Washington, which is pical of the hot interior regions, where apple growing under irrigation has veloped so extensively in recent years and where mildew control has become an portant factor in successful orcharding.

"Pruning.- In both of these investigations it was found important to remove e mildewed twigs in the winter pruning, thus eliminating sources of hold-over fection in the dormant buds and rendering much easier the control by spraying ring the growing season. Mildewed shoots are easily detected during the dormant ason by their gray or silvered appearance. Isolated infections which exist in attered buds, however, make it impossible to control the disease by pruning one. Cutting off infected shoots during the growing season and burning the runings are also a help in establishing control, but in doing so spores are scat-ered and may spread the disease unless the trees are coated with a protective ray.

"Spraying schedule for the control of apple powdery mildew.

"Application and time	Materials
First mildew spray ("pink spray") Apply just before the blossoms open.	Lime-sulphur, 1 to 50; or iron sulphid.
Second mildew spray (calyx spray) Apply as soon as most of the petals have fallen.	Lime-sulphur, 1 to 50; or iron sulphid.
Third mildew spray. Apply about two weeks after the calyx spray	Do.
Fourth mildew spray. Apply about four weeks after the calyx spray.	Lime-sulphur, 1 to 50; or iron sulphid; or ammoniacal copper carbonate; or neutral Bordeaux mixture; or sal soda-Bordeaux mixture.
Later sprays. Apply if necessary at intervals of about three weeks until latter part of August.	Do."

Fisher, D. F. Control of apple powdery mildew. U. S. D. A. Farmers' Bul. 1120. ay, 1920.

References

1) B. M. Duggar. Fungous Diseases of Plants, p. 226.

2) Hesler & Whetzel. Manual of Fruit Diseases, p. 113.

3) Ballard, W. S. & Volck, W. H. Apple Powdery Mildew and its Control in the Pajaro Valley. U. S. D. A. Bul. 120. September 3, 1914.

4) Fisher, D. F. Apple powdery mildew and its control in the arid regions of the Pacific Northwest. U. S. D. A. Bul. 712. October, 1918.

5) H. S. Jackson. Powdery mildew of apple. Ore. Exp. Sta. Crop Pest Report 1911-12, p. 236.

6) Steward, V. B. Apple powdery mildew. Cornell Exp. Sta. Bul. 358: 181. 1915.

7) Fisher, D. F. Control of apple powdery mildew. U. S. Department of Agriculture. Farmers' Bulletin 1120. 1920.

POWDERY MILDEW OF GRAPE

Caused by Uncinula necator (Schw.) Burr.

This disease apparently originated in Japan and was first reported in Europe in 1845 and in America in 1859.

Great losses have been caused in Europe by this disease. In some parts of the United States, also, severe damage has been done.

1. Examine specimens and compare with other mildews studied, especially, the appendages on the perithecia. What is the distinguishing characteristic? Draw a perithecium.

Discussion.

Jackson (3) describes the symptoms as follows:

"Symptoms.- The disease may attack any herbaceous part of the vine. On the leaves the fungus appears in the form of white or greenish white patches of mildew. These may run together till the greater part of the leaf is covered. The fungus may also attack the young canes, beginning at the base in the form of small patches; or in severe cases the whole surface may be covered. The green or white mildew is easily rubbed off, leaving on the canes brownish spots which soon turn black. If severely attacked the canes fail to grow or mature properly. When the disease attacks the blossoms they fail to set. If the young fruit is attacked when quite small the berries may drop off. If attacked when half grown they develop irregularly and the affected parts become hardened, the ripe berries becoming irregular in form. If severely affected they may crack, thus becoming useless for table or market use. If this cracking is early, they may still be used for wine, though in moist seasons they may be attacked by various molds. The berries are usually not attacked after they begin to ripen. When only slightly affected the berries may ripen without cracking, but are disfigured by spots or blotches which reduce their value for market purposes."- Jackson, H. S. Diseases of Small Fruits. Oregon Agricultural College Biennial Crop Pest and Horticultural Report. 1911-12: 269.

Control.- The best known method of combating grape powdery mildew is by the application of sulfur in the form of dust. Bioletti and Flossfeder (4) give the following directions for sulfuring:

"Number of Sulfurings.- The number of times it is necessary to sulfur will depend on the locality, the variety, the season and the sources of infection and will vary from one to six. In the interior valleys two will usually be enough if the work is done regularly and properly in all the vineyards.

"The first sulfuring should be done when the shoots are not more than six or eight inches long. Later, the foliage becomes dense and it is difficult to cover every part of the surface with sulfur. At the first sulfuring it should be possible to see daylight through all parts of the vine.

"The first sulfurings are preventive and are the most important and effective. If we wait until mildew is perceived, the cost of control will be much greater and it will often be impossible to control the disease completely.

"The second sulfuring should be given during or just before blossoming. At this time the interior of the mass of foliage will be dense in places, but the first sulfuring has already covered the parts which might escape the second. With muscats and most wine grapes these sulfurings will usually give perfect control if there are no summer irrigations and no untreated vines or parts of vines in the immediate vicinity. With very susceptible varieties, such as the Carignane, or excessively vigorous growers, like the Emperor, or where summer irrigation is practised, a third sulfuring may be necessary about the time the grapes are half grown. Only imperfect work or abundant reinfection from untreated vines should necessitate more than three treatments except in the coast regions of summer fogs and even here three will usually be sufficient.

"Time of Sulfuring.- The time of day is of no importance, providing there is not so much wind as to prevent proper distribution or so much moisture that the leaves tend to stick together and escape the sulfuring. Dry, moderately warm weather is the best, but the first sulfuring should be done even if such weather does not occur.

"Distribution of the Sulfur.- It should be the aim in sulfuring to eradicate absolutely every vestige of mildew from the vineyard. This can be done only by placing sulfur on every exposed surface. If a single leaf in the vineyard escapes it may produce enough spores to reinfect the whole. This complete distribution is easily accomplished at the first sulfuring if done at the time recommended. It is possible but more difficult, during blossoming and practically impossible later. It is for this reason that the early sulfurings are the most important.

"Proper distribution can be accomplished only with very fine sulfur, which easily floats in the air, and with a distributing machine which throws it out in a spreading cloud. The object should be to produce a cloud of light sulfur dust which will float through the vine and leave the particles of sulfur distributed on every minute piece of surface. This cannot be done with can shakers or sulfur bags, except by the use of unnecessarily large amounts of sulfur. Some form of blower which will thoroughly break up the adhering masses of sulfur particles and expel them with sufficient force is needed. Suitable machines are obtainable in California, but all in use are of foreign manufacture."- Bioletti, Frederic T. and C. C. H. Flossfeder. Oidium or Powdery Mildew of the Vine. California Experiment Station Circular 144. pp. 8-9. December, 1915.

References.

(1) Duggar, B. M. Fungous Diseases of Plants, p. 229.

(2) Hesler & Whetzel. Manual of Fruit Diseases, p. 242.

(3) Jackson, H. S. Powdery Mildew of Grape. Ore. Exp. Sta. Crop Pest Report. 1911-12. p. 269. 1913.

(4) Bioletti, F. R. and Flossfeder, F. C. H. Oidium or powdery mildew of the vine, Calif. Exp. Sta. Cir. 144. 1915.

(5) Bioletti, F. R. Oidium or powdery mildew of the vine. Calif. Exp. Sta. Bul. 186: 315. 1907.

POWDERY MILDEW OF GOOSEBERRY

Caused by Sphaerotheca mors-uvae (Schw.) B. and C.

1. Examine specimens of gooseberry fruits, leaves and twigs which are infested with powdery mildew and compare symptoms and general effect with the powdery mildews previously studied. Mount perithecia and compare with perithecia of the powdery mildews of wheat and of apple and grapes.

References.

(1) Duggar, B. M. Fungous Diseases of Plants, p. 221.

(2) Jackson, H. S. Gooseberry Mildew, Ore. Exp. Sta. Crop Pest Report. 1911-12, p. 264.

(3) Swingle, D. B. Fruit Diseases in Montana. Gooseberry mildew. Mont. Exp. Sta. Cir. 37: 324. 1914.

(4) Stewart, V. B. Some important leaf diseases of nursery stock. The gooseberry mildew. Cornell Exp. Sta. Bul. 358: 200. 1915.

(5) Salmon, E. S. On the present aspect of the epidemic of American gooseberry mildew in Europe. Roy. Hort. Soc. Jour. 29: 102. 1905.

(6) Owens, C. E. Gooseberry mildew control. Oregon Exp. Sta. Crop Pest and Horticultural Report, 1915-20: 152. 1921.

POWDERY MILDEW OF ROSES

Caused by Sphaerotheca pannosa (Fries) Lev.

The fungus causing rose mildew is very closely related to if not identical with, the fungus causing powdery mildew of peach.

1. Examine specimens of diseased leaves, stems and blossom buds. Take notes and compare with powdery mildews previously studied. Make mounts of both conidial and perfect stages of the fungus. Compare with similar stages of mildews of wheat, apply and gooseberry.

References

(1) Duggar, B. M. Fungous Diseases of Plants, p. 224.

POWDERY MILDEW OF PEACH

Caused by Sphaerotheca pannosa, var. persicae.

References

See under mildew of roses, also the following:

(1) Hesler & Whetzel. Manual of Fruit Diseases, p. 314.

(2) Jackson, H. S. Powdery Mildew of Peach, Ore. Exp. Sta. Crop Pest Report 1911-12. p. 257.

(3) Whipple, O. B. Peach Mildew, Colorado Exp. Sta. Bul. 107: 3. 1906.

(4) Stewart, V. B. Some important leaf diseases of nursery stock. Mildew of rose and peach. Cornell Exp. Sta. Bul. 358: 221. 1915.

(5) Stewart, F. C. Peach Mildew. N. Y. (Geneva) Exp. Sta. Bul. 328: 370. 1910.

(6) Ceasar, L. Peach Diseases. Powdery Mildew. Ontario Agr. Dep't. Bul. 201: 40. 1912.

SUPPLEMENTARY STUDY OF POWDERY MILDEWS

Examine specimens of powdery mildew on the leaves of such plants as oak, willow, maple, dogwood, alder, hazelnut, clover, etc. and, after determining the characteristics of the appendages and the number of asci in a perithecium, identify it in the proper genus by means of the following key:

Key to Genera of Powdery Mildews

A. Perithecia contain a single ascus.
   1. Appendages simple, flexuous, and undivided at the tip.....Sphaerotheca
   2. Appendages once or more dichotomously divided at the tip.....Podosphaera
B. Perithecia containing several to many asci.
   1. Appendages never more than slightly swollen at the base.
      a. Appendages simple or more or less flexuous, or irregularly branched, mycelial-like; without tip peculiarities.....Erysiphe
      b. Appendages usually straight, once or more dichotomously branched at the tip.....Microsphaera
      c. Appendages usually straight and spirally inrolled at the tip..Uncinula
   2. Appendages swollen at the base so as to form an enlarged plate or knob.....Phyllactinia.

General Review on powdery mildews.

Describe the vegetative phase of a typical powdery mildew. Where does the ycelium live? How does it get nourishment from the host? What are haustoria? y what means do powdery mildews spread throughout the summer? How do they live ver winter? Describe control measures for each of the powdery mildew diseases tudied. What one fungicide seems to excell all others in effectiveness as a eneral remedy for powdery mildews? What did we find to be the best fungicide or the downy mildews?

How do the powdery mildews rank with the downy mildews in number and seriousess of diseases caused?

EUROPEAN CANKER

Caused by Nectria galligena Bres.

Laboratory study.

a. Symptoms.- Examine cankers, large and small and note effect upon the host. Note the surface of the canker and also the margin. Can you detect any marked difference between specimens of cankers from New York state and those collected in Oregon. Can you offer any suggestion as to the cause of this difference? Find the minute, red fruiting bodies of the fungus. Make habit sketch of canker, Draw fruiting body much enlarged. Compare this canker with the anthracnose canker of apple previously studied as to general appearance and appearance of fruiting bodies of causal fungus.

b. Morphology of the causal organism.- This fungus produces both conidia and ascospores. The conidial pustules are irregular in shape and appearance and when open and shedding spores have a whitish color. The perithecia bearing the ascospores develop on older cankers following the conidial stage. The perithecia are minute flask-shaped structures barely large enough to be seen by the naked eye and may be distinguished by their red color.

(1) Make mounts of both stages, or examine mounts that have been already prepared. Draw.

c. Notes.- Write up complete notes, supplementing the laboratory by reading the discussion or the references below.

Discussion.

The following quotations set forth the most important known facts concerning European canker.

"This disease, troublesome for many years in Europe, was noted on apples in America about 1899. At this time it was found doing damage in Nova Scotia and in

New York State. From time to time reports of the European canker on apple have come from various parts of the United States and Canada, and now it is known to prevail over a wide area on this continent. It has gained most prominence, as an apple disease, in eastern Canada, New York, New England, and the Pacific Coast. The disease is perhaps of European origin. It has been well-known there for scores of years, because of its destructiveness not only to the apple, but also to many forest trees. Previous to 1880, however, no profound investigations were made abroad; about this time considerable attention was given to it in German writings. Subsequently the disease has received more than usual consideration in Italy, Austria, Switzerland, France, Australia, New Zealand, Holland and England.

"In Europe, this canker is the most destructive and most dangerous of all fruit diseases. Thousands of trees are killed in their prime. In many localities certain varieties can no longer be grown, and there are even districts in which apple culture has become a real problem as a result of the ravages of this disease. Nor are the losses confined to the apple nor to fruit trees. It will be less difficult to give an impression of the economic importance of this disease if brief reference to the plants affected is made. Among these are, in addition to the apple, the pear, quince, cherry, gooseberry, lime, beech, maple, ash, alder, hazel-nut, linden, plane-tree, oak, hornbeam, iron-wood, dogwood and magnolia. In Germany, for example, beech-stands are often ruined by it. And while the greatest damage done to forest trees is in foreign countries, its importance on the apple makes it a serious pest.

"In America, its range and destructiveness have not reached proportions which even approach those in Europe. Yet in certain sections, already referred to, the canker is becoming troublesome; and it seems to be on the increase.

"So far as varietal susceptibility is concerned, little information is at hand for American conditions. But German authorities say that growers have always spoken of cankerous varieties. It is held by some that thick-barked varities are more resistant than others. Again, the matter of whether a variety is susceptible or resistant is said to depend directly upon its susceptibility or resistance to frost-injury. Certain writers say that varieties of apples that grow erect and open, remain free from this canker, whereas others near by suffer severely. In contradiction to these observations and opinions, it is held that all varieties suffer alike, and that so-called resistant sorts, when planted among diseased trees, soon exhibit a susceptibility equal to that of their neighbors. Any variety, it is said, which is exposed is just as liable to infection as any other. It is admitted by some that weak-growing varieties show larger cankers, because of their poor vigor, yet this is in most cases not worthy of practical notice.....

"Symptoms.- In later stages it is not at all difficult to detect the presence of the European apple-tree canker, nor to distinguish it from other kinds of cankers. In the younger stages, however, it is likely to be overlooked by the casual observer; and when noticed it is found to be not markedly different in appearance from young cankers of other kinds. The first intimation of the disease consists of a slight browning of the bark in a more or less circular area. This is quickly followed by an intense darkening of the affected tissue. The surface of the canker sinks, leaving the edges prominently raised. In a short time a crevice is evident along the margin of the healthy and diseased bark. The affected tissues are killed, become dry, and slough off, exposing the wood, and at the

margin a callus develops. The enlargement of these cankers is slow; within a year the length of the lesions may not increase more than one-half of an inch. But they increase in size the second year, and even for as many as a score of years after, thus resulting in large cankers.

"Older cankers are recognized as of two sorts; open cankers and closed cankers; and there are various intervening types. These names are very descriptive. In the first kind, the wound is open, the wood is exposed, and a series of dead callus-layers center about a common point. While the edge of this type of canker is raised, there is no distinct swelling, and no gall-like appearance. In the closed canker, the whole presents the appearance of a rough gall. The edges are swollen to the extent that the wound is practically closed and the wood almost covered. It is said that open cankers prevail on those erect and more vigorous growing branches and twigs in the crown of the tree. On the other hand, the closed type predominates on the horizontal or hanging limbs. In the case of open cankers the margin continues to extend. The prevalence of this type of canker on erect branches is explained on the basis that such limbs lack the necessary food supply to overcome the progress of the pathogene. Here the pathogene appears to have the advantage as evidenced by the expansion of the lesion and the failure of the host to check the spread. In the closed cankers the host has the advantage. Along the margins of the older cankers, brillant red fruiting bodies of the pathogene develop. These are easily seen with the naked eye, even though they are never larger than a pin-head.

"The lesions center about a wound, a bud, or the fork of two branches. Such wounds as those caused by hail, insects, pruning and frost are common seats of the injury. Very commonly a stub or twig may be found at the center of the canker.

"Cause.- The European canker is caused by the fungus Nectria galligena. As previously intimated the mycelium lives over from year to year in the diseased bark. In the spring and early summer red perithecia develop in the wound and under favorable conditions discharge their ascospores. Conidial tufts are developed at this time of year also, so that there are two kinds of spores for initiating primary infections. It has been shown that insects are highly important as agents of inoculation; the woolly aphis, for example, is very active in carrying the spores of the fungus. In Europe an outbreak of canker is said to follow closely an unusual prevalence of this insect. It has been estimated that in a single canker 300,000 ascospores are available for dissemination. The spores germinate in a few hours and the germ-tubes enter the bark through wounds or lenticels. Within a week the effects of the fungus are visible. The mycelium, developing from the germ-tube, permeates the bark, the wood and the pith. The attacks are confined chiefly, however, to the bark, where the cortical cells are killed by the fungus. As a result of death, the affected portion of the bark turns brown, the cells collapse, and the canker shows a sunken surface. If the atmosphere is continuously humid, conidial tufts arise from the mycelium. From these tufts, conidia are liberated; they are then carried to other points where new cankers are formed. The mycelium grows more rapidly parallel to the long axis of the limb and hence the canker is the longer in this direction. Where the wood is entered, the mycelium invades the sap-tubes in which it passes up and down. It is believed that at points above and below the canker the fungus again attacks the cortex, this time from within, thus forming a new canker without direct external

noculation. About a year after the canker starts to develop the mycelium forms he red perithecia. These may act as a means of carrying the fungus through the winter."- Hesler, L. R. and H. H. Whetzel. Manual of fruit Diseases. 1917: 125-129.

European Canker on the Pacific Slope.

"...on the Pacific Slope the concentric arrangement of callus rings in old cankers is much less in evidence and usually cannot be found in most cankers caused by Nectria galligena. For some reason, possibly due to climatic or other conditions not yet fully understood, the cankers spread very rapidly, sometimes extending for several inches or even one or two feet in a single season and remain more or less closed. One can readily imagine that this continual lateral and longitudinal growth without callus formation can be possible only in a locality where there is a prolonged humid season with moderate temperature such as usually prevails for several months on the Pacific Slope of the Northwest. This perhaps is the reason that we find in western Oregon these large cankers covering many square inches of bark surface without the conspicuous formation of calluses.

"For the most part there is every indication that infections take place at small, sappy, pruning cuts or in crotches. On the Red Cheek Pippin apple and Bosc and D'Anjou pear, cankers have been observed frequently to start in the crotches of the largest branches..... In many instances where there are no pruning cuts there are indications of infection in the crotches of fruiting and leaf spurs. Wherever the bark of the host has become devitalized, through sun scald or winter injury, for instance, the fungus obtains an easy access and from there spreads to the healthier tissues.....

"Although the life history of Nectria galligena should be traced definitely for the humid conditions under which it is found here there are some observations which have already been made which we believe should be presented at this time. Near Corvallis the sappy, soft cankers upon which the macro-and microconidia occur have been observed on D'Anjou pear during the early part of June.....

"Upon cankers caused by N. galligena and which we suspect were formed the same season the senior author has found pustules of the multi-septate, sickle-shaped macroconidia as well as the ellipsoid, one- and two-celled microconidia. These were found on apple cankers collected Febrary 3, 1920 and on pear cankers collected October 13 of the same year..... The microconidia and macroconidia to date have been found in early spring and lasting at least until October. The stromatic cushions of conidia first appear very near the seat of infection and later break out as pustules further out from this point as the canker increases in size."- Zeller, S. M. and C. E. Owens. European Canker on the Pacific Slope. Phytopathology 11: 465-468. November, 1921.

Review.

How can European canker be distinguished from other canker diseases studied? What canker diseases have been previously studied? Give the life history cycle of European Canker. Discuss control measures.

References.

(1) Duggar, B. M. Fungous Diseases of Plants, p. 242.

(2) Hesler & Whetzel. Manual of Fruit Diseases, p. 125.

(3) Brooks, Chas. European apple canker. New Hempshire Exp. Sta. Bul. 144: 128. 1909.

(4) Morse, W. J. The European Apple Canker in Maine. Me. Exp. Sta. Bul. 223: 23. 1914.

(5) Hesler, Lex R. Apple cankers and their control. N. Y. (Cornell) Exp. Sta. Cir. 28. 1915.

(6) Hasselbring, H. Canker of Apple Trees. Illinois, Exp. Sta. Bul. 70. 1902.

(7) Zeller, S. M. and C. E. Owens.- European Canker on the Pacific Slope. Phytopathology 11: 464. Nov., 1921.

SCAB (FUSARIUM - BLIGHT) OF WHEAT AND OTHER CEREALS, and CORN ROOT, STALK AND EAR ROT.

Caused by Gibberella saubinettii (Mont.) Sacc. and Fusarium spp.

1. Laboratory study.

a. Symptoms.- Examine seedlings and also heads and culms of wheat and the other cereals and also corn seedlings and roots, stalks and ears of more mature corn plants. Note the symptoms as illustrated by the various specimens. Read the quotation below and then try to find illustrations of all the symptoms there described.

b. Morphology of the causal organism.

(1) Conidial stage.- Search the surface of the diseased parts for a pinkish fungous growth. Scrape off some of this growth and mount it for microscopic study. You should find conidiospores in your mount. They are long curved spores with one or more cross walls, typical of the form genus, Fusarium. (See the chapter on Fungi Imperfecti). Draw.

(2) Perfect stage.- Examine old specimens of diseased plants, especially old corn stalks for the black perithecia scattered over the surface. Mount some of these and crush out the asci and ascospores. Draw.

2. Text discussion.

Read the following quotations and any other references available and become familiar with the symptoms, life history and control of this disease, especially the relation between the wheat scab and the corn root, stalk and ear rot, Atanasoff (1) gives the main facts of symptoms and life history:

"The cereal crops--wheat, spelt, rye, and oats--and also some grasses are subject to attack by a number of fungi belonging to the genus Fusarium, of which the most common and most important is known, in its ascigerous form, as Gibberella saubinetti (Mont.) Sacc. The organism attacks each of the hosts named above in at least two different ways, producing two distinct pathological conditions. The first condition results from an attack on the root systems and the bases of the young and later of the grown plants, occasionally causing partial or entire wilting The second condition results from an attack upon some of the parts above ground. This may be a rotting of the nodes, found on rye, wheat, and barley, or blighting of the heads of wheat, spelt, rye, barley, and, less commonly, of oats and certain grasses. In all cases the various attacks on the same host are independent of each other. A wheat plant may be attacked underground, or on the head only or on both the roots and the head, and in some cases even on some of the nodes; but in all cases these infections are quite independent.

"Up to the present time little attention has been given to these two forms of attack by Fusarium, and they have commonly been considered two different diseases caused by one or more unknown species of Fusarium.

"However, the results of the work reported here prove conclusively that these two conditions are only two different phases of the same problem.... While G. saubinetii is unquestionably the cause of headblighting of the cereal crops under most conditions and throughout the greater part of this country, it is equally true that under certain conditions and in some parts of the country other species of Fusarium are also responsible for the head-blighting of cereal crops. The following organisms besides G. saubinetii have been isolated from blighted wheat, rye, oats, and barley heads or plants; Fusarium avenaceum (Fr.) Sacc., F. herbarum (Corda) Fries, F. culmorum (W.G. Smith) Sacc., F. culmorum (W. G. Smith) Sacc. var. leteius Sherb., F. arcuosporum Sherb., F. scirpi Lamb et Fautr., F. solani (Mart. pr. p) Ap. et Wr., F. arthrosporioides Sherb., and F. redolens Wr. These species, while very seldom responsible for the headblighting of cereals, are not so unimportant in the rootrot problem of these crops. Indeed, some of them (F. herbarum, F. avenaceum, F. culmorum, and F. culmorum var. leteius) have, in my observations, proved to be as important as G. saubinetii in causing rootrot of the cereal crops.....

"Geographic Distribution.

"The Fusarium-blight of cereals is more or less common thruout the central and eastern cereal-growing sections of the United States. It has been reported by the Plant Disease Survey of 1917, 1918, and 1919 from the following States: Maine, New Hampshire, Vermont, Massachusetts, Connecticut, New York, Pennsylvania, New Jersey, Delaware, Maryland, West Virginia, Virginia, North Carolina, South

Carolina, Georgia, Alabama, Tennessee, Kentucky, Ohio, Indiana, Michigan, Illinois, Wisconsin, Minnesota, Iowa, Missouri, Oklahoma, North Dakota, South Dakota, Montana, and Oregon..... "It has been reported from various parts of Canada....."

"In Europe the disease has been found in England, France, Italy, Germany, Austria, Holland, Denmark, Norway, Sweden, and Russia. In Russia the disease is common throughout the wheat-and rye-growing sections. In Asia it is very common in the Usurian provinces on the Siberian Pacific coast. It has also been reported from Australia."

"Economic Importance.

"The Fusarium-blight of the cereal crops injures the plants in several ways and is generally considered an important disease of these crops. It lowers germination of the seed and causes dying off or weakening of the young seedlings. Later it causes dying and wilting of fully grown plants, and finally it blights the heads, wholly or in part, thus preventing them from filling. The severity of the headblighting varies from a fraction of 1 per cent to 100 per cent, and the loss due to decrease in yield in individual fields and localities may vary from 0 to over 50 per cent."

"If the estimate of the Plant Disease Survey approximates the actual loss due to the blighting of the wheat heads in the States reporting, then the total annual loss for the United States is probably close to 20,000,000 bushels."

"Blighted Seed.

"Wheat kernels obtained from heads blighted or partly blighted by Gibberella saubinetii show marked evidence of the effect of the Fusarium attack and can be easily distinguished in a sample of grain, even when only a very small percentage of such kernels are present. Wheat seed from blighted heads exhibits one of three more or less distinct and definite pathological symptoms, depending upon the time of head infection.

(1) Kernels from heads infected early in their development, possibly during or shortly after the blossoming period, are small in size, being sometimes hardly two-thirds as long as the normal. They are pale greenish gray in color, badly shrunken, not firm, and very light in weight. As a rule, such kernels are never able to germinate. They may be heavily infected or even covered with the mycelium of the fungus if they developed near the point of infection, or they may be perfectly free from any fungus mycelium, if they have developed far above the point of infection where the food supply was cut off.

(2) Kernels from heads infected two or three weeks after the blossoming period may attain nearly a normal size, but they usually have a slightly shrunken appearance. They are grayish white or cream-white in color, soft and starchy in texture, and much lighter in weight than the normal kernel. In this case, also, they may be infested and even covered with mycelium, which is especially evident in the

groove, or they may be entirely free from mycelium, depending on their position in the head with relation to the point of infection. The percentage of germination of kernels in this class is very low.

(3) The third class of kernels consists of those which have been infected shortly before or just after the head is ripe. Such kernels differ very slightly from the normal, except that they are partly discolored, pinkish spots being not uncommon on them. While it is true that Gibberella saubinetii is the most common cause of pinkish red coloring on kernels in all three of these classes, it must be remembered that other fungi, Macrosporium and Alternaria for instance, may in some cases cause this coloring of grain. Kernels of this last class usually germinate normally, but before the young plant has reached the surface of the soil, or before it attains any considerable size, it not uncommonly wilts and dies as a result of infection from the kernel. In many cases, however, the seedling survives the attack and reaches full development.....

"Seedling-Blight.

"Seedlings from seed naturally or artificially infected with Gibberella saubinetii are subject to attacks by this organism at a very early stage of their development, and the visible symptoms of the infection may become evident at the time of the germination of the seed or only a few days later....."

"Footrot.

"Careful examination of the underground portions of winter crops early in spring and of spring crops somewhat later in the season shows partial rotting of the roots, the bases, and, in some cases, the interior of the stems just above the bases. Various fungi may be found associated with this condition on the cereal crops, among which Gibberella saubinetii and species of Fusarium are common.....

"On Stems of Grown Plants.

"Occasionally full-grown plants are killed by Gibberella saubinetii or by one of several Fusarium species just before or shortly after the time of blossoming. The fungus attacks the roots and the stem close to the ground, the first node usually being involved in the infected area. The part of the stem in contact with the ground and the roots below are rotted and are commonly pink or yellowish brown in color. This rotting of the base interferes with the water and food supply of the plant, and wilting of the entire plant is the result.....

"Blighting of Heads.

"Wheat.- The symptoms and effects of headblighting of different varieties of wheat are, in general, the same. The blighted head usually takes on the normal color characteristic of the ripe head of that variety or a slightly lighter color.

"Blighting of the wheat heads can be detected with absolute certainty at a very early stage, three of four days after infection has taken place, provided that weather conditions have been so favorable as to enable the parasite to establish itself on the host and to begin its work of destruction.

"The symptoms of blight infection as they appear on Marquis or some other of the beardless varieties are as follows: The very first sign of blight infection is a slightly brown and water-soaked spot, 2 to 3 mm. in length, on the glumes. The veins appear more water-soaked and have a much darker olive-green appearance than the area between them. The points at which the infected glume or glumes are attached to the rachis soon show the water-soaked appearance also. The water-soaked area increases more or less rapidly, depending on weather conditions, until the whole spikelet is covered. It then spreads to the neighboring spikelets.

"If the weather is dry the infection may remain restricted to one spikelet. At this time the glumes and the spikelets originally infected gradually begin to lose the water-soaked appearance, dry up, and take on the typical color of the ripe head of the particular variety. This drying up of the infected spikelets follows closely the advancing infection, which usually proceeds downward.....The healthy part of the head above the point of infection usually dries up and dies without passing through the water-soaked stage, because of the cutting off of the water and food supply by the fungus at the point of infection.,....."

"The point of infection, even when the attack is in an advanced stage, can easily be located, especially if the weather has been favorable. It is usually covered at first with a short, cottony, slightly pinkish fungous growth, while the rest of the infected area remains free from such a growth. Later, if the weather is favorable, this growth extends farther over the infected area and becomes the substratum on which a layer of conidia develops..... The conidial masses, which were originally slightly pinkish, now become dark salmon to grenadine in color, depending on the causal organism."

"Rye.- The symptoms of headblighting of rye are very much like those of wheat, except that the water-soaked appearance is not so prominent....."

"Barley.- The symptoms of blight on barley heads are usually different from those on wheat and rye, seldom resembling those on the latter. Usually only one kernel is killed, or occasionally several kernels in one row. In some cases the three kernels forming a spikelet are attacked and later, if conditions are favorable, the rest of the head is blighted. The first sign of infection is a small, water-soaked, somewhat brownish spot appearing at the base or the middle of the glume or on the rachis. The water soaking and browning spread in all directions from the point of infection, soon including the whole glume, the whole spikelet, or several spikelets, but the infection is by no means as uniform as it is in wheat and rye."

Oats.- The symptoms of headblighting of oats resemble those of wheat. Because of the structure of the panicle, however, the infection is usually restricted to one spikelet and is therefore not so conspicuous as it is in wheat or rye."

"Life History of Causal Organism...

"Production of Spores.

"conidia.

"Production of conidia upon the host plant is more or less common in all forms of Fusarium attacks on cereals. In many cases it may be so abundant that it leaves no doubt as to the real source of inoculum for subsequent infection in nature.

"On Seedlings.- When a wilted seedling is pulled out and portions of its partly decayed kernel or of the young stem are examined under the microscope, a great number of normally developed conidia can frequently be seen. In rare cases masses of conidia are also formed on the rotted stem above the ground...."

"On Nodes and Bases.- Formation of conidia on the heads of cereal crops, especially of wheat and rye, shortly after infection takes place is common and so abundant as to give them a very distinct pinkish or salmon color....."

"On Dead Organic Matter.- Old straw and pieces of stems and cornstalks in fields where the year before the crop has been heavily infected with the disease were often found to show large pinkish areas bearing numerous conidia, some of which belonged to some of the species of Fusarium which were found parasitizing wheat and corn....."

"Ascospores.

"Whenever the cause of the disease is one of the species having a perfect stage, as is the case with Gibberella saubinetii, the perithecia of this fungus are produced in great number on all infected parts, but especially on the pseudoplectenchymatic structures, on which there has been more or less formation of conidia. Perithecia are formed on seedlings and infected kernels (observed only under greenhouse conditions), on the straw and the heads of the various cereal crops, and on the stalks, sheaths, and ears of corn. The ascospores play an important role in the life of this organism, since they are likely to resist extreme weather conditions and furnish inoculum for the first infection in the spring.....

"Source of Natural Infection.

"An important source of infection is the seed used for sowing. Cereal seeds carry, externally, viable conidia of Gibberella saubinetii, as well as of Fusarium spp., and many of the kernels are internally infected with these fungi.....

"Overwintering of the Fungus.

"The organism, because of its comparative resistance to cold and drying, overwinters in various ways. When introduced into the soil with the winter crops, it overwinters in the form of mycelium and conidia where these are formed on the

killed seedlings and on other organic substances. It also overwinters in the form of mycelium in and on the seed, straw, heads, and cornstalks that have been infected with the fungus the summer before. The organism has been isolated from such plant parts kept out of doors throughout the winter and spring. During the winter of 1918 it was frequently isolated from cornstalks fed to the cattle on the University farm and from cornstalks that had been taken out into the fields with the manure or for cattle feeding.

"The mycelium of the organism present in infected straw and heads of wheat, rye, and barley when stored in the laboratory at room temperature and moisture was found viable after 12 months. In the infected seed it remains viable even after the second year.

"The undeveloped perithecia of the organism, which are often found in the fall on the straw and heads of the cereal crops, on cornstalks and sheaths, and on many grasses are another form in which this organism overwinters. In the spring these perithecia mature and form numerous ascospores, which are later liberated from the perithecia and carried to the various susceptible hosts. Mature ascospores in perithecia on wheat heads and cornstalks preserve their viability for over 8 months when kept in the laboratory at room temperature and moisture."- Atanasoff, D. Fusarium-blight (Scab) of wheat and other cereals. Jour. Agr. Res. 20: 1-15. Oct. 1, 1920.

Relation of Wheat-scab to Corn root-rot.

Hoffer, Johnson and Atanasoff (2) found that a close relationship exists between corn root, stalk and ear rot on the one hand, and wheat scab on the other. The chief points in their findings are quoted below.

"Field observations have shown a conspicuously greater abundance of wheatscab in fields where the wheat (Triticum aestivum) was grown immediately following corn that was infected with the Fusarium-rot of the root and stalk. This was especially true in Shelby County, Indiana, where wheat, according to a common practice, was sown in standing corn.

"A similar condition was also noted in Dane County, Wisconsin, this summer, where spring wheat was grown immediately following a corn crop. Both in Indiana and in Wisconsin under these conditions abundant development of perithecia of Gibberella spp. was found on the old cornstalks remaining in these fields. These perithecia were mature and well filled with viable ascospores at the time when the wheat, in all cases observed, was in head.

"Water suspensions of these ascospores both from Indiana and from Wisconsin cornstalks gave positive results when used as inocula on wheat heads.... The appearance of the infected heads thus artificially inoculated was identical with that of wheat heads naturally infected with scab.

"Cultures from Gibberella spp. on old cornstalks have also proved virulently parasitic on the roots of corn plants....

"Similar results on both wheat and corn have been obtained by using cultures from naturally infected wheat heads....."

Control.

Quoting again from Hoffer, Johnson and Atanasoff (2) we find the following:

".....it seems evident that, in order to lessen the losses from these diseases on corn and wheat, it is necessary to recognize this intercrop parasitism and develop field practices accordingly. In general, the use of the best-adapted, disease-free seed on clean soil should be practiced.... A crop rotation avoiding wheat following diseased corn is undoubtedly important, unless the cornstalks can be cut close to the ground, removed, and the remaining stubble plowed under before the wheat is planted. Badly scabbed wheat should not be used for seed. Ordinary seed treatments will not control wheatscab hence, only clean seed on carefully prepared clean soil should be used."

Further recommendations for the control of corn root, stalk and ear rot are given by Holbert and Hoffer (3).

"Control of corn root, stalk and ear rot.

"Select well-matured seed at the proper time from healthy plants. Avoid all leaning or broken plants and all broken ear shanks even if the ears look healthy.

"Select about five times as many seed ears as will be needed, to allow for discarding later all ears found to be diseased.

"Cure and store the seed ears in a dry, well-ventilated place. Full directions are given in Farmers' Bulletin 1175.

"After the seed ears are thoroughly dry, discard all ears with denting that is too rough or with pink, discolored, cracked, or shredded shank attachments, or with moldy, discolored, or starchy kernels.

"Make a special germination test of ten representative kernels from each seed ear selected.

"Keep for seed only those ears represented on the germinator by ten healthy seedlings, cutting open the ten kernels to make sure they are not rotted, even if the sprout appears to be healthy...."

References.

(1) Atanasoff, Dimitr. Fusarium-blight (Scab) of wheat and other cereals. Jour. Agr. Res. 20: 1. Oct. 1, 1920.

(2) Hoffer, G. N., A. G. Johnson and D. Atanasoff. Corn root rot and wheat scab. Jour. Agr. Res. 14: 611. Sept. 23, 1918.

(3) Holbert, James R. and Geo. N. Hoffer. Control of the root, stalk and ear diseases of corn. U. S. D. A. Farmers' Bul. 1176. 1920.

(4) Manns, T. F. and J. F. Adams. Corn root rot diseases. Delaware Exp. Sta. Bul. 128. 1921.

(5) Hoffer, George N. and J. R. Holbert. Selection of disease-free seed corn. Purdue Exp. Sta. Bul. 224. 1918.

(6) Koehler, Benjamin, J. G. Dickson, and J. R. Holbert. Wheat scab and corn rootrot caused by Gibberella saubinetii in relation to crop successions. Jour. Agr. Res. 27: 861. March 15, 1924.

ERGOT OF GRAINS AND GRASSES

Caused by Claviceps purpurea (Fr.) Tul.

This is one of the most common diseases found on certain grains and grasses, and while it probably does not as a rule greatly reduce yields, yet in certain cases where pasture or forage crops are badly infested a serious disease is sometimes caused in stock which eat quantities of the ergots (sclerotia) formed from infected ovaries of the grain or grass.

1. Examine specimens of various plants infested with ergot. Note the long black bodies (sclerotia) which protrude from the head where healthy seeds should be found. Draw head to show sclerotia.

2. Life History.

a. In early stages of infection before sclerotia appear a sticky exudate appears at the point where later a sclerotium appears.

b. In the early stage of development, conidia are formed on the diseased ovaries.

c. After the mature sclerotia fall to the ground and lie there over winter, the fruiting bodies of the perfect stage appear. They are stalked bodies with rounded heads in which are cavities containing asci. See the chart and specimens of these fruiting bodies (stromata) in vial. Draw sclerotium with stromata attached, also section of stroma enlarged to show cavities (perithecia) containing asci. Draw one sac with spores, enlarged.

3. Notes.- Write up complete description of such features as have been learned in laboratory. Read the references and complete notes. Give especial attention to the economic loss due to ergotism in cattle. Note also that ergot has a certain medicinal value. What is this value?

References

(1) Duggar, B. M. Fungous Diseases of Plants, p. 244.

(2) Stevens & Hall. Diseases of Economic Plants, p. 358.

(3) Stevens, F. L. Fungi which cause plant disease, p. 21.

(4) Coulter, Barnes & Cowles. Text-Book of Botany. Vol. I, p. 77.

(5) Curtis, C. C. Nature and Development of Plants, p. 23.

(6) Heald, F. D. & Peters, A. T. Ergot and Ergotism. Neb. Exp. Sta. Press Bul. 23. 1906.

(7) Salmon, D. E. Enzootics of ergotism. U. S. D. A. Report 1884: 212-252.

(8) Freeman, E. M. Minnesota Plant Diseases, p. 128.

(9) Ericksson, J. Fungoid diseases of plants, p. 126.

(10) Williams, Thomas A. Some plants injurious to stock. Ergot. S. Dakota Exp. Sta. Bul. 33: 38. 1893.

(11) Johnson, A. G. & Vaughan, R. E. Ergot in rye and how to remove it. College of Agr. Univ. of Wis. Ext. Service Cir. 94. 1918.

(12) Seymour, Edith K. and F. T. McFarland. Losses from rye ergot. Phytopathology 11: 285. July, 1921.

BLACK KNOT OF PLUMS AND CHERRIES

Caused by Plowrightia morbosa (Schw.) Sacc.

This is a very common and destructive disease in the eastern and central United States. In Oregon, it occurs commonly on wild cherry. The following hosts are listed for various parts of America: cultivated sour cherry, wild red and yellow plum, chickasaw plum, choke cherry, wild red cherry and wild black cherry.

1. Symptoms.- Examine knotty branches and describe symptoms. Make habit sketch.

2. Morphology of causal organism.- Examine the surface of the black stroma. Note the projecting perithecia. Can you find an opening (ostiolum) on the top of any of the perithecia? Examine a section of the stroma. Is it composed of fungus tissue or host tissue? Note the cavities of the perithecia. Draw. Mount sacs and spores under the microscope. Draw.

References.

(1) Duggar, B. M. Fungous Diseases of Plants, p. 248.

(2) Hesler & Whetzel. Manual of Fruit Diseases, pages 180 and 356.

(3) Beach, S. A. Black knot of plum and cherry. N. Y. (Geneva) Exp. Sta. Bul. 40: 25. 1892.

(4) Hesler, Lex R. Diseases of the plum. Black knot. N. Y. Agr. Dep't. Bul. 79: 1190. 1916.

BLACKROT CANKER OF POMACEOUS FRUITS.

Caused by Physalospora cydoniae Arnaud.

1. Laboratory Study.

a. Symptoms:

(1) On fruits.- Examine fruits in the early and in advanced stages of decay. Note color of skin and flesh in different stages. At what stage do fruiting bodies of the fungus appear? Describe?

(2) On branches.- Examine young and old cankers. Draw and describe. Does the fungus fruit on the cankers?

(3) On leaves.- Draw a leaf showing the characteristic spots.

b. Morphology and life cycle of causal organism.

(1) Conidial stage.- Examine under the microscope sections thru the pycnidia on both twigs and fruit. Draw. Describe the pycnidium and the conidiospores (pycnospores).

(2) Perfect stage.- If the perfect or ascigerous stage is available examine it as for the conidial stage. Draw section of the perithecium showing asci and ascospores. Describe the life history. (See references).

2. Text discussion.

Hesler (34) gives the following rather full discussion of the Black-rot disease of apples and other pomaceous fruits.

Hosts.

"The black rot, leaf spot, and canker of pomaceous fruits is primarily a disease of the apple, Pyrus Malus L. It affects other trees also, however, especially the pear (Pyrus communis L.), the quince (Cydonia vulgaris Pers.), and the crab (Pyrus coronaria L.), showing on these hosts symptoms similar to those on the apple. In addition the pathogene infests the dead parts of a great variety of other trees and shrubs, but in such cases there is usually no evidence that it has been the causal factor in the death of the tissues. In the State of New York, at least, this disease is of economic importance only on apple trees.

"Varietal susceptibility.

"Of fruit to black rot.- The summer varieties of apples are affected by black rot at the time of ripening, while winter varieties commonly suffer in storage. In Connecticut black rot is a most troublesome storage rot (Clinton, 1915: 5).

"Of foliage to leaf spot.- Brooks and DeMeritt (1912: 183) (2), in New Hampshire, note striking differences in the varietal resistance of apple seedlings to leaf spot. In Virginia, Ben Davis and Black Twig are more severely attacked than are other varieties (Reed, Cooley, and Rogers, 1912: 5) (3). Salmon (1907) (4), writing from England, states that among the varieties most affected there are Peasgood, Nonsuch, Cox's Orange, and others. The writer has noted that Chenango, Baldwin, Rhode Island, and Twenty Ounce show spotted foliage more commonly in New York than do other varieties.

"Of limbs to canker.- Varieties of apples exhibit a marked difference in susceptibility to the disease, and this variation is not the same with respect to the different parts affected. In western New York Twenty Ounce is the variety most severely affected by the canker. This variety is rarely found unaffected by canker, even in orchards that are managed according to improved methods. Neglected trees of the Twenty Ounce variety are often killed. Paddock (1899 b: 181) (5), says that certain growers in New York State have noted that Twenty Ounce is most likely to be attacked. He lists other varieties in order of their susceptibility, as follows: Baldwin, Wagener, Rhode Island, Tompkins King. Regarding the Esopus, Paddock (1899 b: 180) (5) says that this variety has apparently run out because of this disease. In Ontario, Ben Davis, Northern Spy, and other varieties are severely injured by the disease (McCready, 1910) (6).

"Names

"The disease on the fruit is called black rot, ring rot, blossom-end rot, and brown rot. The first name is descriptive and definite, and seems desirable.

"Lesions on the foliage are termed leaf spot, and frog-eye.

"The names apple canker, black rot canker, and New York apple tree canker, are most commonly applied to the disease when it occurs on the bark. The term

apple canker needs some qualification, since many kinds of cankers occur on the apple. It was under such an exigency that Paddock (1899 b: 180, footnote) (5) first used the name New York apple tree canker. The writer prefers to use this name regardless of its length, not alone because of priority, but also because of general usage.

"History.

"The black rot of the fruit.- The disease on the fruit was observed by Peck (1879: 20) (7) in Schoharie County, New York, and he quotes the remark of stage-coach passengers that they "never before knew of apples rotting on the tree". Arthur (1885) (8) records black rot of quince fruit at Geneva, New York, and notes the importance and infectious nature of the disease. Scribner (1890) (10) notes the disease in New Jersey and describes the symptoms and causal nature. The same year Baccarini (1890) (9), in Italy, observed the rotting of otherwise sound apples, pears, and peaches, the decay occurring in a storeroom of fruit gathered when ripe; this was a new appearance of the disease in that country. Two years later Halsted (1892) (11) discussed the black rot of quince in New Jersey. Sturgis (1893, a and b) (12) notes the disease on quinces in Connecticut in August, 1892, and later (1894) confirms the work of Halsted. Kinney (1895 c) (13) figures and describes the disease in Rhode Island. In the same year it became an object of control measures in Kentucky (Garman, 1895) (14). Black rot of apple is reported by Burrill and Blair (1901) (15) in Illinois; they give a careful description of the disease and distinguish it from other apple rots.....

"The leaf spot.- The earliest record which the writer has found concerning the leaf spot is that by Alwood (1892) (16), who reports brown spot of apple foliage in Virginia. He had noticed it for several years but states that it was not serious until 1891, when an outbreak occurred over the State. Kinney (1895 b) (17) describes the disease in Rhode Island on apple and pear leaves, and says that it was very common in that State in 1895. Stewart (1896) (18) reports having found the disease on Long Island in May, 1895: he notes that it had been observed in 1894 at Westbury, Long Island, by Professor Beach. Lamson (1899) (19) lists the disease from New Hampshire and reports it as having done serious damage in some cases. Corbett (1900) (20) reports "brown spot", or "frog-eye", as common throughout the southern and eastern counties of West Virginia, and as being more injurious than either blight or scab.....

"The canker form.- According to Paddock (1899 b: 180) (5), orchardists have been familiar with this canker disease for years. Attention was first called to the injury by Waite (1898 a) (22), in a paper read at the meeting of the Western New York Horticultural Society in 1898. In the Spring of 1898, Paddock (1898, a and b, and 1899 a) (21), began investigations which extended through two seasons. He records having noticed the disease as early as 1891, and says that it became important about 1895. In 1897, according to Paddock (1899 b: 192) (5), considerable "twig blight" was found at Odessa, New York... .....

"Geographical Occurrence

"The disease has a very general distribution throughout the temperate regions of the globe. Heretofore it has been regarded as a disease peculiar to eastern

and middle western America, but it is now apparent that its limit is no longer to be so regarded. It has been found in Canada, particularly in Ontario, (Bethune, 1909) (23), Quebec (Lochhead, 1905 and 1909) (24, 25), and Nova Scotia (Plant Pathology Herb., Cornell University Exsicc. no. 2657, and Gussow, letter to the writer). It occurs in Europe from Italy to England, according to the observations of Shear (1913: 81-82) (26).....

"So far as the writer has been able to ascertain, the disease is widely distributed in America.....The canker form is commonest in the East, especially in New York, while fruit rot seems to occur more frequently in the New England and Middle Atlantic sections, although Wilson (1913) (27) reports its occurrence throughout North America east of the Rocky Mountains. In the Middle Western States, particularly Ohio and Indiana, the pear and quince, as well as the apple, appear to have suffered considerably. Quaintance and Scott (1912) (28) state that the leaf spot occurs in all sections east of the Rocky Mountains where the apple is grown.....

"Economic Importance

"General considerations.- The nature of the losses caused by this disease makes very difficult the possibility of an estimate concerning them. So far as the writer has found, no reliable data are at hand in regard to this point. The combined injuries produced by the canker, the black rot, and the leaf spot are doubtless greater than is commonly supposed. It is undoubtedly true that the New York apple tree canker is often confused with other cankers by some growers, thus increasing the difficulty of obtaining reliable estimates on the distructiveness of this disease.....

"Nature of Losses.

"Injury to fruit.- Whenever pome fruits are attacked they are rendered worthless so far as their market value is concerned. The extent of injury may be small while the fruit still hangs on the tree, but ultimately in storage complete destruction is likely to result.....

"Injury to foliage.- The damage to foliage depends on the extent of the infection. In the milder cases the injury is not appreciable. In the more severe cases, partial or even complete defoliation may occur from six weeks to two months before the ripening of the fruit, as a result of which the fruit either drops from the tree or remains small and is of poor quality. In such cases the fruit buds are so weakened as to decrease the possibility of a crop the following year, and the vitality of the trees is impaired.....

"Injury to limbs.- As a rule the canker is confined to the orchard, although Wilson (1913) (27) says that young nursery stock may be killed. Bothune (1909: 28-29) (23) reports that cankers cause a great amount of damage in Ontario, Canada. J. W. Eastham (letter to the writer), of the Ontario Experiment Farm, states that this is the most prevalent canker in the region east of Toronto, and according to Brooks (1909) (29) it is the commonest canker in New Hampshire. Warren and McCou Brooks (1905: 341) (30) refer to this form of the disease as causing more loss in Wayne County, New York, than any other disease except scab. They report it as very

serious in fourteen per cent of the orchards, and as doing considerable damage in nineteen per cent of them. In Niagara County, New York, Cummings (1909: 304) (31) found canker affecting the orchards as follows: slightly, sixty-one orchards; considerably, sixty-three; seriously, thirty-seven. Paddock (1899 b: 181) (5) cites a case in which, in an apple orchard of 80 acres, the trees on 30 acres were ruined and had been taken out; the trees on the remaining 50 acres were then of little value.....

"Symptoms.

"On the fruit.- The disease on the fruit is primarily a ripe rot, but it may appear several weeks before maturity of the fruit. It may begin anywhere on the surface or at the blossom end. Frequently the lesions are centered about an injury such as that caused by insects or hail.

"Usually there is only one spot on a fruit. The skin at first becomes brown in a small area, but later darkens, finally turning black. On green fruit the affected part may turn black before enlarging to any great extent, whereas on fruit that is ripe or ripening, the whole may be involved before it darkens appreciably. Often concentric bands of uniform breadth and of slightly different shades of color appear about the center of the lesion. The affected area is distinct from the healthy part, and the diseased tissues are not of unpleasant taste as in many fruit decays. Later stages in the development of the rot show a shriveled and much wrinkled surface, which typically becomes covered with black pustules. These characters may be assumed within a month or in less time. Ultimately a dry mummy is produced, which may hang to the tree for a year or more.....

"On the leaves.- The foliage of the apple is more frequently attacked than that of the pear and the quince. The spots usually appear about the time the leaves open.....

"The number of lesions that may appear on a single leaf varies from one to several, and these may be scattered or localized on the surface. The first evidence of a spot is a minute purple speck, which soon enlarges until it has reached a diameter of from two to ten millimeters, averaging about four millimeters. The purplish color is maintained for a considerable time, during which the margin is somewhat indefinite. Later the lesion is of yellowish brown color and the spot assumes a more or less circular shape, while the margin becomes more definite. Still later the margin becomes elevated and the diseased area sunken. As the spots grow older they become lobed, due to a secondary extension of the pathogene from one or more points, and finally a series of more or less concentric areas produces an irregular blotch in which the outline of the original spot can be recognized. The center has now become grayish brown, the entire lesion presents an appearance which has given rise to the name frog-eye.....

"On the limbs.- In western New York, young spots may be found on the bark at any time from the last of April until toward the close of the growing season. Numerous young cankers have been observed on Twenty Ounce trees in Monroe County orchards during the month of August. It is the rule that the larger limbs are much more susceptible than the twigs, and the trunks show comparatively few lesions.

The cankers on the trunks occur more or less uniformly on the southwest side of trees. The limbs more commonly show the diseased spots on the upper side. Lesions are very often found about the base of a small limb or about a wound in the bark.

"In the earlier stages of the formation of a canker, the bark is slightly sunken and reddish brown in color. The diseased area slowly increases in size and darkens, and, although not conspicuous at a distance, the spot is readily distinguished from healthy tissue on closer examination. Some lesions remain very small, measuring only a few centimeters in their longer diameter; in such cases the canker usually dies out at the end of the year. Where the injury is larger, the diseased spot enlarges from year to year for a distance of a meter or even more.

"It is often observed that a canker is merely a superficial roughening of the bark. In other cases the bark is killed to the wood and becomes conspicuously cracked.

"The discolored area may extend over a considerable surface; or, regardless of its size, a crevice may appear at the margin, limiting, temporarily at least, the extent of the lesion. Further spread of the pathogene results in the formation of a prominent spot, which soon forms a second line of demarcation between the healthy and the diseased tissue. Repetition of this process from one or more points at the margin occurs, thus producing a lobed appearance; or, the spreading may arise from all points about the first marginal crack, so that a series of concentric crevices is developed, as described for frog-eye of the leaves. The bark remains closely appressed to the wood for at least a year; later the dead bark cracks and falls away, exposing the wood and a callus around the margin of the wound.....

Morphology of causal organism.

"Perithecia.- The perithecia have been found by the writer on the twigs of apple. (Pyrus malus L.) and of witch-hazel (Hamamelis virginiana L.), and have been described by him in another publication (Hesler, 1913: 293) (32). Recently the writer has found the ascogenous form on twigs of white oak (Quercus alba L.)

"The perithecia are usually scattered, standing separate from one another. Sometimes, however, from two to four fruit bodies are joined together, but no stroma has ever been observed. They are buried in the cortical tissues, protruding at maturity by a short, papillate ostiole. Their form is globose to subglobose, measuring from 180 to 324 μ in the vertical diameter by 300 to 400 μ * in the horizontal diameter, averaging about 225 by 325 μ .....

"The asci, with the interspersed paraphyses, usually fill the cavity of the perithecium..... They are usually clavate, although they sometimes tend to be cylindrical, measuring from 21 to 32 μ by 130 to 180 μ .....

The ascospores are ellipsoidal, or often inequilateral; they measure from 10.8 to 15.2 μ by 23.4 to 34.2 μ , averaging 11.5 by 28 μ . They are hyaline to greenish yellow.....

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* μ = microns.

"The number of spores in each ascus is typically eight, but exceptions have been found in the fungus on apple twigs.....

"Pycnidia.- The morphology of the pycnidial form of this fungus is variable. The pycnidia are situated in the cortical tissues and are usually scattered and distinct, although on the same organ of the host they may be single, confluent, or united into a stroma. The number of pycnidia per unit of area is usually less on woody substrata than on fruits; on apple fruit there may be from one hundred and twenty to one hundred and fifty pycnidia per square centimeter.

"The typical simple pycnidium measures from 200 to 300μ in each diameter, whereas the compound fruit body may vary from 200 to 460μ in the vertical diameter and from 200 to 720μ in the horizontal diameter. Their shape is in general the same as that of the perithecia, globose to subglobose.....

"The pycnidia arise from the mycelium and are found abundantly on cankers and decayed fruit.....

"Pycnospores.- The morphology of the pycnospores has been more carefully studied than that of the pycnidia. In the same and in different pycnidia on the same and on different plants, wide variation with respect to size, color, shape, and septation have been observed.

"The average mature pycnospore measures about 12 by 15μ, although the range in single or in different pycnidia on the same or on different hosts or host parts may be considerable. From the measurement of hundreds of pycnospores, it has been found that they range from 7 to 16.2μ broad by from 16 to 36μ long, while the averages from different hosts range from 9.5 to 13.3μ by 19.8 to 27.8μ .....

"The mature pycnospores are brownish, varying from a very light to a dark ferruginous color. The color darkens with age, so that the very youngest mature spores are hyaline.....

"The typical pycnospore is ellipsoidal, globose, or somewhat elongated forms...

"A pycnidium may contain only one-celled spores or only two-celled forms, or both kinds may be present. Occasionally three-celled and even four-celled pycnospores have been observed.

"Life History Studies.

"The fungus passes the winter as mycelium in the tissues of the host and as pycnospores in pycnidia. If a canker is examined in the spring when growth is resumed by the host plant, the margin of the old lesion may show discoloration. The writer has frequently planted bits of the bark from the edge of a canker in agar plates, pure cultures resulting. This is evidence of the resumption of growth of the mycelium in the old lesion. It is stated by Caesar (1909) (33) that as a rule the fungus does not die out but continues to extend in every direction year after year, finally girdling the limb. In this connection Paddock (1899 b: 189) (5) writes as follows: 'In some instances the mycelium apparently lives over winter

and continues its growth the following spring. The formation of the largest cankers can scarcely be explained in any other way.'

"It is a common thing to find, in the winter and spring, pycnidia bearing mature spores on mummified fruits. These fruiting bodies may be observed also on cankers and on fallen twigs, bark, and leaves. In western New York cankers and dead twigs are the most important sources of the inoculum.....

"The rôle of the ascosporic stage in hibernation is not certain. As previously indicated, the perithecia are rare on Pyrus malus in New York, and it would seem, therefore, that the ascospores from this source are of relatively little importance in initiating infections. Whether this stage is common on other plants in this State, and important as a source of inoculum, are matters remaining to be investigated.....

"Time of spore discharge.- The time of the year when spores are disseminated may be approximated by noting the date of the first appearance of the diseased spots, and by keeping careful watch of the behavior of the pycnidia in the field....

"From observations made by the writer it seems that the date of first discharge in the spring varies with the season. Apparently the ostiole does not open at a temperature below 60°F. (15.5°C.) or in the absence of moisture, and a period of humidity of several hours duration is necessary. The dates for spore discharge observed at Byron, New York, are May 16, 1912, and May 12 and 23, 1913. In 1911 spores were found coiling on July 17, but the process must have occurred earlier. That they continue to be liberated throughout the summer is shown by the appearance of new infections on foliage from May until September 20, 1913. In 1911 several young spots were found on bark in the middle of August. Ripe and green fruit infections also show that spores are disseminated in August and September.....- Hesler, L. R. Black rot, leaf spot, and canker of pomaceous fruits. Cornell Univ. Agri. Expr. Sta. Bul. 379, August, 1916.

Control.

The following discussion of control measures is quoted from Hesler and Whetzel (35).

"The treatment of this disease consists in the eradication of cankers and in protecting the susceptible parts by the use of fungicides.

"The treatment of cankers may be followed along one of two lines. The affected limb may be cut from the tree, or the diseased bark may be removed. The grower must study the problem and must proceed in accordance with the conditions. The size and value of the limb and the extent of the infection will be criteria. Small limbs, that is, those with a diameter less than one inch, should in all cases be removed. It would rarely be profitable to attempt to treat a canker on a branch of this size. If the larger cankered limb is valuable because of its producing powers, then the size of the canker must finally determine the course to pursue. Smaller cankers on large productive limbs can be profitably removed. If the lesion extends more than half around the limb, no attempt should be made at removing the canker; the entire branch should be removed.....

"The effectiveness of spraying for canker is a question frequently raised. As a preventive this operation is worthy of attention; as a cure it is out of the question, for once the fungus is in the bark the spray material cannot reach it.....

"For the control of black-rot of apples, bordeaux mixture is said to be more effective than lime-sulfur. Apply bordeaux 4-4-50 as follows: (1) about July 15, when the disease is just appearing, and (2) two weeks later. As a matter of precaution against damage in storage, avoid injuring the fruit in any way. Spraying for leaf-spot should be done according to the schedule outlined for apple-scab...." Hesler and Whetzel. Manual of Fruit Disease. 1917. pp. 52-56.

References.

(1) Clinton, 1915. Notes on plant diseases of Connecticut. In Report of the Botanist for 1913. Connecticut (New Haven) Agr. Exp. Sta. Ann, rept. 38 (1914): 1-29.

(2) Brooks, Charles, and DeMeritt, Margaret. 1912. Apple leaf spot. Phytopath. 2: 181-190. pl. 17. fig. 1-6.

(3) Reed, H. S., Cooley, J. S., and Rogers, J. T. 1912. Foliage diseases of the apple. Virginia (Polytech. Inst.) Agr. Exp. Sta. Bul. 195: 1-25. fig. 1-13.

(4) Salmon, E. S. 1907. Apple leaf-spots. Gard. chron. ser. 3: 42: 305-306, Fig. 120-124.

(5) Paddock, Wendell. 1899 b. The New York apple-tree canker. New York (Geneva) Agr. Exp. Sta. Bul. 163: 177-206, pl. 1-6.

(6) McCready, S. B. 1910. Black rot canker (Sphaeropsis Malorum Peck). Ontario Agr. Coll. and Exp. Farm. Ann. rept. 35: 41-42.

(7) Peck, C. H., 1879. Report of the Botanist. New York State Mus. Nat. Hist. Ann. rept. 31: 19-60.

(8) Arthur, J. C., 1885. Quince rot. In Report of the Botanist. New York (Geneva) Agr. Exp. Sta. Ann. rept. 3: 372.

(9) Baccarini, P., 1890. Note patologiche. Ital. nuovo giorn. bot. 22: 64-70.

(10) Scribner, F. L., 1890. Black-rot of the apple. Fungus diseases of the grape and other plants and their treatment, p. 81-83. fig. 1606.

(11) Halsted, B. D., 1892. The black rot of the quince. In Some fungous diseases of the quince fruit. New Jersey Agr. Coll. Exp. Sta. Bul. 91: 8-10, fig. 5-6.

(12) Sturgis, W. C., 1893 a. Black-rot (Sphaeropsis Malorum Peck). In Common fungous diseases and their treatment. Connecticut (New Haven) Agr. Exp. Sta. Bul. 115: 6-7.

(13) Kinney, L. F., 1895 c. The brown rot. In Horticultural Division. Rhode Island Agr. Exp. Sta. Ann. rept. 7: 192-193. fig. 12.

(14) Garman, H., 1895. Spraying experiments in 1895. Kentucky Agr. Exp. Sta. Bul. 59: 111-129. Illus.

(15) Burrill, T. J. and Blair, J. C., 1901. Apple fruit-rots. Illinois Univ. Agr. Expr. Sta. Circ. 37: (12 pages, unnumbered). Illus.

(16) Alwood, W. B., 1892. Brown spot. In Four diseases of the apple, and treatment of same. Virginia Agr. and Mech. Coll. Agr. Exp. Sta. Bul. 17: 59-62.

(17) Kinney, L. F. 1895 b. The leaf spot of the apple and pear. In Horticultural Division. Rhode Island Agr. Exp. Sta. Ann. rept. 7: 188-189, fig. 7-8.

(18) Stewart, F. C., 1896. A new leaf-spot disease of apples. In Report of the Mycologist. New York (Geneva) Agr. Exp. Sta. Ann. rept. 14 (1895): 545-546.

(19) Lamson, H. H., 1899. Apple diseases. In Notes on apple and potato disease. New Hampshire Coll. Agr. Exp. Sta. Bul. 48: 146-147.

(20) Corbett, L. C., 1900. Brown spot, frog-eye. In Fruit diseases and how to treat them. West Virginia Agr. Exp. Sta. Bul. 66: 202-204, fig. 2.

(21) Paddock, Wendell., 1898 a. An apple canker. Science n. s. 8: 595-596.
1898 b. Additional notes on an apple canker. Science n. s. 8: 836-837.

(22) Waite, M. B., 1898 a. An apple canker. Western New York Hort. Soc. Proc. 43 9-11.

(23) Bethune, C.J.S., 1909. Fungus diseases and insect pests. Also, Cankers on apple trees. Ontario Agr. Coll. and Exp. Farm. Ann. rept. 34: 26-32.

(24) Lochhead, William., 1905. Black rot canker. Ontario Agr. Coll. and Exp. Farm. Ann. rept. 30: 49, fig. 4-5.

(25) Lochhead, William., 1909. Apple diseases. In Fungus diseases in Quebec in 1908. Quebec Soc. Prot. Plants from Ins. and Fung. Dis. Ann. rept. 1: 31.

(26) Shear, C. L., 1913. Some observations on phytopathological problems in Europe and America. Phytopath. 3: 77-87.

(27) Wilson, G. W., 1913. New York Canker. In Notes on three limb diseases of apple. North Carolina Agr. Exp. Sta. Ann. rept. 35: 47-49, fig. 1.

(28) Quaintance, A. L. and Scott, W. M., 1912. Apple leaf-spot. In The more important insect and fungous enemies of the fruit and foliage of the apple. U. S. Agr. Dept. Farmers' Bul. 492: 35-36, fig. 20.

(29) Brooks, Charles., 1909. Black rot, canker and leaf spot. In Some apple diseases. New Hampshire Agr. Exp. Sta. Bul. 144: 123-126, fig. 20-24.

(30) Warren, G. F. and McCourt, W. E., 1905. The apple-tree canker. In An apple orchard survey of Wayne County, New York. Cornell Univ. Agr. Exp. Sta. Bul. 226: 341-345, fig. 86-87.

(31) Cummings, M. B., 1909. Apple orchard survey of Niagara County. Cornell Univ. Agr. Exp. Sta. Bul. 262: 277-320, fig. 26-40.

(32) Hesler, L. R., 1913. Physalospora Cydoniae. Phytopath. 3: 290-295. Illus.

(33) Caesar, L. 1909. Black rot canker of apple trees. In Lime-sulfur wash. Part II. Practical and popular treatment. Ontario Agr. Dept. Bul. 177: 59-61, fig. 16.

(34) Hesler, L.R. Black-rot, leaf-spot and canker of pomaceous fruits. N. Y. (Cornell) Exp. Sta. Bul. 379. 1916.

(35) Hesler and Whetzel. Manual of Fruit Diseases. 1917. pp. 45-56.

(36) Zeller, S. M. Sphaeropsis malorum and Myxosporium corticola on apple and pear in Oregon. Phytopathology 14: 329. July 1924.

APPLE SCAB

Caused by Venturia inaequalis (Cooke) Winter.

Apple scab is probably the most wide spread of all apple diseases in this country. In Oregon it certainly is more widely known and attracts more attention than the anthracnose disease, its nearest rival. As far as immediate or direct damage is concerned, scab is undoubtedly the most serious apple disease in this state although in some neglected orchards anthracnose causes more permanent injury.

1. Laboratory Study.

a. Symptoms.

(1) On fruit.- Examine samples of young and mature fruits affected with scab. Note appearance of new infections and of older scabs. Note broken and loosened cuticle around margin of scab. Note corky growth over diseased surface in old lesions.

(2) On leaves.- Observe scab-infested leaves and describe appearance. Do the spots on the leaves resemble those on the fruits? What is the difference if any?

(3) On twigs.- Examine twigs infested with scab and compare appearance with affected fruits and leaves.

(4) On flowers.- Note that petals, sepals and ovaries of flowers are sometimes attacked at blossoming time.

b. Pathological histology.- Examine, under the microscope, sections cut through a typical scab on fruit. Compare with sections of normal apple tissue. What is the difference in the cell structure just beneath the epidermis?

c. Morphology and life history of causal organism.

(1) Conidial stage.- In a section of a mature scab find the layer of fungus tissue at the surface. Note the row of conidiophores and the conidia which they bear. Draw a narrow strip across the section to show fungus and host tissue. Be sure to show the fruiting structure in detail.

(2) Perfect Stage.- On old dead leaves, collected from the ground in early spring find, with the aid of hand lens or binocular microscope, minute black fruiting bodies of the fungus breaking out through the epidermis of the leaf. These are ascus-bearing fruiting bodies (perithecia) which develop saprophytically on the dead leaves from mycelium which lives over winter in leaves which were infected with scab the previous summer. Examine, under the microscope sections of dead leaf cut through perithecia to show sacs and spores. Draw. The spores are forcibly ejected from the perithecia during or following spring showers and infection takes place on blossoms, young leaves or young fruits.

d. Notes.- Write a complete account of apple scab. Supplement the facts learned in the laboratory with additional data obtained from reading the references. Give particular attention to control measures as related to life history.

2. Field work.- If conditions permit, study this disease in the orchard.

a. Find the perfect stage on old, dead leaves.

b. Observe new infections on flowers, leaves or young fruits.

c. Estimate percentages of infection.

d. Bring specimens into laboratory, and examine to see if spores are present.

Discussion.

"Wallace (3) has given a very full and comprehensive review of this disease. Extensive quotations from his article follow.

"The disease, which is known as scab, black spot, scurf, or the fungus, and which has in some cases been called rust, occurs on the leaves and the fruit, and occasionally has been found on the twigs, of the apple. The name "scab" is used almost exclusively in the United States and is the name that will be used in this account.

Historical.

"The scab disease apparently exists in every country where apples are grown. It was reported early in the nineteenth century by Fries (1819) from Sweden, and some years later by Wallroth (1833) from Germany. The first authentic record from America is by Schweinitz (1834), who reports scab on Newton Pippins in New York and Pennsylvania. The disease was first noticed in England in 1845, according to Berkeley (1855), and in Australia in 1862, according to McAlpine (1902). Its introduction into Australia was attributed to a Seckel pear imported from America;

this is doubtful, however, since the scab disease of the pear has since been shown to be entirely distinct from that of the apple.

Symptoms.

"On the leaves.- The scab is likely to appear earliest on the lower side of the leaves. The diseased area usually appears first as an olive discoloration slightly darker than the normal surface of the leaf. The color deepens with age until dark brown or black is reached, the spot having a more or less velvet-like appearance. As noted by Aderhold (1896), on the lower side of the leaf there is a tendency for the lesion to extend along the veins and the midrib and to diffuse irregularly and indefinitely into the healthy area; whereas on the upper surface of the leaf the lesion appears first as a slight olive-green discolcration of a lighter shade of green than the healthy surface of the leaf but dull and some what velvety. The natural luster characteristic of the upper surface of the leaf is destroyed. The spots may be few and scattered; or they may be so numerous as to coalesce, coating almost the entire surface. The diseased areas may be distinctly bordered or they may spread out irregularly and indefinitely into the healthy part of the leaf.....

"Later the scab spots become darker, changing to brown and finally, in some cases, to nearly black. In some cases the natural form of the leaf is not destroyed; in other cases some distortion results. Very often the diseased surface protrudes, forming a convex surface with a corresponding concavity on the opposite side of the leaf. In time the tissue under many of the diseased spots is killed outright, forming dead areas, which often crack as shrinkage occurs. The condition is most common near the close of the season. In extreme cases considerable defoliation may result.

"On the fruit.- The lesion on the fruit usually appears from the first as a darker-colored spot than is produced by the disease on the leaves. Sometimes the spots are almost black when first visible, or they may be dark olive, changing to reddish brown or black. The spots are usually very small at first and they enlarge more slowly than do those on the leaves. They are more sharply bordered on the fruit than on the leaves.

"As the scab spot on the fruit grows older its appearance changes markedly. The central and older part becomes bare, brown, and corky, while the margin is black. A more or less whitish band, due to the loosened cuticle, mayssurround the black margin. Sometimes the scab spots may enlarge so as to cover rather large areas and cracking of the fruit results, due to excessive loss of moisture content from the underlying unprotected tissues.

"Scab spots resulting from late autumn infection differ somewhat in appearance from those developing early while the fruit is very young. The spots as they first appear are more dense and black. Often they will have enlarged considerably before the cuticle is ruptured. They seldom reach the stage described above, in which the center becomes bare and brown.

"On the twigs.- The disease rarely occurs on the twigs, at least in many localities. Affected twigs have, in general a scurfy appearance. The bark becomes blistered and later ruptured in places, presenting an appearance similar to scabby pear twigs, which are very common.....

General Estimates of Loss.

"McAlpine (1902) estimated the annual average loss due to apple scab in Victoria, Australia, at 40,000 pounds (approximately $194,000), which is equivalent to $48.50 an acre. Stevens and Sherman (1903) state that one grower reports an increase of $1000 in value of his crop due to spraying, at an outlay of $125 to $150; and that in the State of Illinois apple scab is estimated to have caused $6,000,000 damage in one year, or sixty per cent of the total loss through all enemies. Marlatt and Orton (1906) state that the loss fromscab amounts to many million dollars each year. Scott and Quaintance (1907) estimate that scab often affects fifty to seventy-five per cent of the fruit over wide areas, and not infrequently causes total failure of the crop by killing the young fruit when in blossom or soon after. Gossard (1908) estimates a net profit of $3 to $7 for each tree, due to spraying for control of insects and disease, at a cost of 30 to 50 cents for each tree.

"Estimates of loss in New York.- Some interesting figures in regard to the importance of scab disease, as shown by the gain due to spraying apple orchards,.... Warren (1905) reports an average gain of $47 an acre, in 1904, in orchards sprayed three times over those not sprayed.....

"..... It was found that orchards having no scab to 5 per cent of scab gave an average income of $143 an acre, with a yield of 382 bushels; while those having 76 to 100 per cent of scab gave an average income of only $88 an acre, with a yield of 248 bushels. This shows an apparent loss of $55 an acre where scab was abundant.....

"Cummings (1909) made similar estimates in his survey of Niagara county. Here unsprayed orchards gave an average yield of 261 bushels with an average income of $45 an acre, while three sprayings resulted in a yield of 577 bushels and an increase of $126 an acre.....

"..... Cummings found one fifth of the orchards in Niagara county unsprayed. Since that is one of the most progressive fruit-growing counties in New York, there is no doubt that it would be a conservative estimate to say that one fifth of the orchard area of the State is unsprayed, making a total of 75,274 acres on which a loss of $47 an acre would be suffered. This gives $3,537,878 as an estimate of the annual loss to this State through neglect of spraying. This estimate represents only the loss where no spraying is done. Many thousands of acres are ineffectively sprayed, and if the loss on these could be estimated it would add much to the aggregate.

"Figuring on a similar basis for the United States, it is found in the same census that there were 201, 794,764 apple trees on 5,044,869 acres. One fifth of this acreage gives 1,008,973.8 acres of unsprayed orchard, on which the loss at $47 an acre would amount to $47,421,768.60.....

"Nature of the loss.- Ordinarily the reduction in quality of scabby apples is considered to be the main cause of loss. This, while it is important, is only one of the several factors. It will be shown later that early scab infection, if not controlled, in some years almost entirely prevents the setting of fruit; it also very materially reduces the size of individual apples, while a single lesion retards growth on the affected side and causes unsymmetrical development. The unmistakable dwarfing effect has been apparent in experimental work during two epidemic years of apple scab, when it was noticed that apples from unsprayed trees were uniformly smaller than those from sprayed trees. Green (1891) determined that there was a loss of fifty per cent on scabby fruit as compared with sound fruit.

"A fourth important factor of loss to be considered is the effect on the keeping qualities of the fruit. This effect is indirect, in that the disease furnishes a point of entrance for Cephalothecium roseum, the pink-rot organism, Penicillium expansum, the brown ripe-rot fungus, Sphaeropsis malorum, the black-rot fungus, and other organisms.

"In addition to the immediate effect on the crop of the current year, there is doubtless, in case of severe leaf infection a devitalizing effect on the tree as noted by Bailey (1895, pp. 13-14). To some degree this prevents the formation of fruit buds for the following year and hinders the normal wood growth which is the basis for future crops.

"Morphology.- The apple-scab disease is caused by the fungous pathogene Venturia inaequalis. The mycelium of the fungus is in some cases hyaline when young, but it soon becomes tinted and varies from olivaceous to reddish brown. It is septate, branches very irregularly, and is subject to modifications as influenced by environment, varying particularly in the different stages of the life cycle of the fungus. The hyphae vary from 3 to 5 μ in diameter in the living host, but in the dead leaves they may be as much as twice this size. In the living host the mycelium will be found, in the early stages on leaves, confined to a region between the cuticle and the epidermal cells; on the fruit the epidermal cells are attacked and usually destroyed.

"Previous to the formation of conidia this subcuticular mycelium divides into one or more layers of densely packed, rounded, and at first hyaline cells (stroma), from which the conidiophores are produced. The outer layers later take on a brownish tint. On the leaves this stroma may consist of only one layer of cells, but more than one layer is likely to occur over the veins or the firmer parts of the leaves. On the fruit the number of layers is much greater, due apparently to the fact that the thicker cuticle offers a greater resistance which is not overcome until the time for the formation of more cells has elapsed. This resistance is apparently overcome partly by means of a solvent action of the fungus, and partly by the pressure exerted which finally pries the cuticle loose. The conidiophores that arise from these cells are reddish brown in color and while young are somewhat ovate in form. They have an inner hyaline wall which protrudes from the apex to form the spore. At first the spores are hyaline and are rounded in a sac-like manner at the free end, but later they become reddish olive-colored and are mostly lanceolate but somewhat irregularly so and variable in form. They are mostly unicellular in size, measuring 12 to 22 μ in length by 6 to 9 μ in breadth. The form and general appearance of the conidiophore changes with its age and the number

of conidia that it has produced. The older conidiophores assume a more or less irregular form, showing a distinct knee and a change in the axis of growth at the point where each spore was borne. They are usually unicellular, but sometimes old conidiophores become septate.

"The perithecia are imbedded in the tissue of the leaf, usually protruding sufficiently to form a small dome-shaped pimple which is sometimes large enough to be easily visible to the naked eye but at other times only discernible by the aid of a lens. They are spherical or subspherical, with a somewhat beak-like projection at the ostiole. Six or more simple tapering bristles.... sometimes surround the ostiole. The perithecial wall is dark olive-green to brown in color, with polygonal reticulations. The asci are oblong to clavate, often somewhat curved,..... without paraphyses. The ascospores are olive-brown, two-celled, with the upper cell somewhat broader than the lower,..... There are eight ascospores in each ascus.

Life History.

"Perithecial stage.- It is well known to pathologists that Venturia inaequalis has two distinct stages in its life cycle.....

"The perithecia appear most abundantly on the exposed surface of the leaf as it lies on the ground. They are often not easily discernible with the naked eye, but appear under a lens as small, dome-shaped pimples on the surface of the leaf. They are sometimes confused with the fruiting bodies of other fungi which are very similar in external appearance to the perithecia of Venturia and which are frequently more abundant than the latter. However, by one who is familiar with their appearance they can usually be distinguished from other forms on examination with a hand lens; the pimples commonly have more of a dome-like form and are plumper than those of other fungi that are likely to be confused with them.

"Development of perithecia.- The perfect stage of the fungus begins to develop in fall or early winter. After the scab-infected leaves have fallen and decay has set in, the mycelium, which during the summer does not penetrate deeper than the epidermis, permeates the entire leaf tissue and sometime during the fall or winter begins to form perithecia.....

"Clinton (1901) found signs of perithecial formation in sections of fallen leaves made as early as October, and could occasionally connect these perithecia by mycelial thread with the subcuticular mycelium. He notes that the perithecia usually occur on the lower side of the leaf, especially along the veins, and believes that they usually originate from a late infection on the lower side of the leaf. In the writer's observations during the past three seasons, perithecia have been found to occur abundantly on both sides of the leaf. They seem to develop mostly on the side that faces upward as the leaf lies on the ground. There appears to be a negatively geotropic tendency.

"Time of maturity of ascospores.- In nature the ascospores usually begin to mature at or about the time when the apple blossoms are ready to open. In 1908 mature spores were found on May 4 and in 1910 on May 1; the blossoms were about

:o open in each..... The ascospores do not all mature at one time and the ·ipening process may continue for about one month.....

"The development of the two forms of this fungus as outlined by Clinton (1901) agrees in general with the writer's observations as recorded in the text. The following paragraphs are quoted from Clinton's bulletin:

May. Scab first appears on young apple leaves and fruit and during this month and June obtains its greatest foothold.

July to September. The warm, generally dry, weather is not very favorable for spreading the disease to the leaves, and fruit usually suffers but little from further infection.

September to October. Scab appears to develop somewhat more abundantly and especially on the lower surfaces of the leaves, but not necessarily in vigorous fruiting condition.

October and later. On the fallen dead leaves there are signs of the formation of the perithecia of the Venturia stage.

October to April. Perithecia slowly develop as weather conditions prove favorable.

June. Venturia stage disappears.(See Childs (11) on this point).

"Late infection and scab development in storage.- While the cycle indicated above may be regarded as the usual one, it is to be noted that seasonal variations markedly influence the development of the disease. In the case of abundant fall rains accompanied by fog, or the occurrence of excessive dew, a late infection of scab appears. The disease may not appear on infected fruit until the fruit is stored. This phenomenon has been called to the writer's attention recently by several investigators and growers. It is not new, however, for Goethe (1889) noted that scab developed in certain cases and new infections occurred after the fruit had been stored.....

"How the fungus passes the winter.- Several possibilities have been suggested in answer to this question. One suggestion is that conidia which may lodge on the twigs or about the bud scales are able to retain their vitality and to germinate when favorable conditions arise in spring. Another suggestion is that the stroma of the fungus on twigs, or even on decayed leaves or fruit, may withstand the winter and produce in spring a new crop of conidia to start the infection.....

"Considering all the evidence, the writer is convinced that the ascigerous stage is the principal agent involved in carrying the fungus over winter. It has been shown that twig infection is not of very common occurrence and that the conidia cannot withstand winter temperatures. Furthermore, evidence that the ascospores are the chief source of primary spring infection is furnished by the fact, noted more in detail in another part of the bulletin, that orchards the leaves of which have been plowed under or burned early in spring or late in fall are less seriously affected than are orchards in which the leaves are left exposed.

Varietal Susceptibility.

"Perhaps the best method of obtaining data as to varietal susceptibility in ase of this disease is to summarize the results that have been obtained and the bservations that have been made by various investigators for years past. In eviewing this work it becomes evident that too much stress should not be laid on single set of observations. It is found that certain varieties may be resistant n one year and susceptible in another year under conditions which for average arieties are as favorable to the disease in the one case as in the other. The xperiments reported by Aderhold emphasize this point. The writer, on comparing is own results with those previously reported by others, finds the above statement o hold good.

"Baldwin, for example, is usually listed among resistant varieties; yet on he check trees in the writer's experiments during 1910 Baldwin trees yielded inety-eight per cent of scabby fruit, much of which was badly infected. In 1909, hich was not an epidemic year, the Baldwins were as badly diseased as were the hode Island Greenings. The Ben Davis, which is usually reported as resistant, as as badly scabbed during both seasons as was the average variety.

"It may be possible that the reputation of these varieties is due partly to heir color. The scab is not so conspicuous on the dark-colored fruit as on that f the light-colored varieties. Many growers who had very scabby Baldwins in the eason of 1910 considered them clean, while Rhode Island Greenings, which were eally less diseased, were considered a scabby lot. However, as previously stated, t is possible that Baldwin and Ben Davis may be more or less resistant in some easons, since it has been proved that such variations do occur.

"There seems to be no satisfactory explanation for such variations, although several suggestions may be offered. They may be due to a relation of the weather conditions or of the time when weather favorable to infection occurs, to certain stages in the development of the fruit and the leaves of certain varieties. For example, a Baldwin may be just at its most susceptible stage at the time when weather favorable to infection occurs in this season, and in the next season may have passed this stage; while a Rhode Island Greening may so develop with respect to the weather as to give the opposite results each year. In some varieties the susceptible period may be reached sooner or may last longer than in other varieties, this explanation, however, is as yet only theoretical.

Control.

"Sanitary Measures.- It should not be inferred from what has been said regarding infection by the perfect stage of the scab fungus that the disease can be controlled by sanitary measures alone. There will probably always be enough fallen leaves exposed to permit some infection, from which further spread may be rapid. It is doubtless true also that spores can be carried for considerable distances from neighboring orchards. If, however, the dead leaves are turned under, it is probable that much less danger will result from primary infection usually occurring about blossoming time. In most cases the primary attack would probably be so light as to cause no serious results aside from furnishing convenient sources for more

bundant secondary infection. Theoretically, then, it should be possible to dis-
ose of many sources of primary infection by plowing the orchard in the previous
utumn, or early in the spring before the ascospores have ripened.....

"Pruning is an important factor. The denser the foliage of the tree, the
ore slowly it dries out after each rain. The longer the tree remains wet, the
etter is the opportunity for spores of the fungus to germinate and cause in-
ection.

"Selection of Resistant Varieties.- This practice offers very little promise
s a means of control. In the discussion of varietal susceptibility, it has been
hown that no variety is likely to remain immune, or even very resistant, for many
ears. Further, even though a resistant variety could be obtained, the grower
ould not afford to sacrifice other desirable qualities for this one. Therefore it
ould seem better to select for quality of fruit and for productiveness, vigor, and
ardiness of tree, and depend on some method of protecting the tree from scab.

"Spraying.- In view of the above discussion it will become evident that the
ost important method to be employed in controlling this disease is by timely
pplications of a protecting substance to the host.....

"The problem is to find the best fungicide and to learn how and, still more
important, when it should be applied.....

"Spraying fallen leaves.- The question is sometimes asked, will the spray
falling on the dead leaves beneath the tree kill the ascospores or prevent them
from being discharged? A study of the mechanism by means of which the spores are
discharged will answer this question. The spores, being borne in a closed
perithecium....., are protected from the fungicide until their discharge takes
place. It will be seen further, ... that the asci containing the spores protrude
beyond the surface of the leaf, passing the spores safely through any coating of
spray material that may be present without even necessitating their contact with
it."- Wallace, Errett. Scab Disease of Apples. Cornell University Agricultural
Experiment Station Bulletin 335. pp. 546-590. September, 1913.

Spray schedule for apple scab.

In Oregon for the average season a maximum of five sprayings is recommended.

1. The delayed dormant.
This is applied soon after the winter buds open, when the leaves on the
fruit spurs are one-third to three-fourths inch long. Blossom buds just appearing
but not yet separated.

2. "Pink" spray.
So named because at this stage the individual blossom buds are showing
pink color, although not yet open.

3. "Calyx" spray.
Just after most of the petals have fallen but the sepals are not yet
closed.

4. Ten-day spray
Applied ten days after the "calyx" spray.

5. Thirty-day spray.
Applied thirty days after the "calyx" spray.

If the grower is expert enough in judging weather and other conditions he may omit at least the last of these five sprays providing there are no early season infections in the orchard. For absolute safety in the average season, however, the entire schedule should be adhered to strictly.

Relation of period of Ascospore Discharge to Spray Schedule.

Childs (11) gives the following facts on this point.

"Mature ascospores were first ejected at Hood River on March 20, 1916. At Corvallis, Oregon, this activity was first noted on February 25. In both localities apple trees were largely dormant.

"The last ejection of ascospores occurred at Hood River on June 27. At Corvallis abundant discharge was observed as late as May 20, after which date no examinations were made.

"The study of ascospore discharge demonstrates beyond a doubt that the delayed-dormant application given while the leaves are small and undeveloped cannot be safely dispensed with in the Hood River and Willamette Valleys, at least since the ejection of ascospores begins before the foliage has even started. This would probably also hold true in all sections of Western Oregon and Washington,..... It is not at all unlikely that other sections could also be included except for our present limited knowledge."- Childs, Leroy. New Facts Regarding the Period of Ascospore Discharge of the Apple Scab Fungus. Oregon Agricultural Experiment Station Bulletin 143. p. 11. May, 1917.

Relation of height of fruit on tree to scab infection

"In large trees a much greater percentage of scabby apples was found on the upper portions of the trees than nearer the ground.

"At least a quarter of the crop, in the case of fifteen-year-old trees, was found to be produced at a greater height than fifteen feet. Nearly half of this, the best quality fruit produced on the trees, was found to be scabby.....

"The average wind velocity prevailing in Hood River during the spraying season prevents the accomplishment of good work from the ground at a greater height than 22 or 23 feet. In the case of trees as large as those studied, both fruit and foliage in the upper 8 feet of the tops are only partly covered, and in many applications missed entirely. From the infection which takes place here, rains wash down millions of spores onto the fruit and foliage below, which, if not completely protected, become infected. Protection becomes gradually less from the ground to

he top of the tree, while the chances of late infection occurring, are much reater from the top to the bottom, owing to the proximity of the summer spores resent in the tops of the trees.

"For older orchards, the installation of up-to-date spray machinery fitted ith three leads of hose and a tower, together with the cutting back of long out-f-the-way limbs, will facilitate spraying and cut down the chances of top infec-ion."- Childs, Leroy. Observations on the Relation of the Height of Fruit to pple Scab Infection. Report of the Hood River Branch Experiment Station for 1916. regon Agricultural Experiment Station Bulletin 141. pp. 16-17. February, 1917.

Review.

State the geographical distribution of apple scab in the U. S. Discuss susceptible and resistant varieties. Describe the symptoms on all parts of the plant. Give details of life history, paying particular attention to perpetuation, dissemination and infection. Discuss scab infections in relation to weather conditions. Discuss control measures in detail.

References.

(1) Duggar, B. M. Fungous Diseases of Plants, p. 264.

(2) Hesler & Whetzel. Manual of Fruit Diseases, p. 3.

(3) Wallace, E. The scab disease of apples. N. Y. (Cornell) Exp. Sta. Bul. 335: 545. 1913.

(4) Reddick & Crosby. Dusting & Spraying experiments with apples. N. Y. (Cornell) Exp. Sta. Bul. 369: 308. 1916.

(5) Jackson, H. S. Apple scab. Ore. Exp. Sta. Crop Pest Report 1911-12. p. 238.

(6) Lawrence, W. H. Apple Scab in Eastern Washington. Wash. Exp. Sta. Bul. 75. 1906.

(7) Jackson, H. S. & Winston, J. R. Control of Apple scab. Ore. Exp. Sta. Hood River Branch Report 1913-14. p. 6.

(8) Childs, Leroy. Observations on the relation of the height of fruit to apple scab infection. Ore. Exp. Sta. Hood River Branch Report. 1916.

(9) Lawrence, W. H. The apple scab in Western Washington. Wash. Exp. Sta. Bul. 64. 1904.

(10) Barss, H. P. & Lovett, A. L. Orchard spray program for 1919. Ore. Extension Bul. 224. 1919.

(11) Childs, Leroy. New facts regarding the period of ascospore discharge of the apple scab fungus. Ore. Agr. Exp. Sta. Bul. 143: 11. May, 1917.

(12) Barss, H. P. Apple Scab and Pear Scab. Ore. Agr. College Extension Cir. 160. 1922.

PEAR SCAB

Caused by Venturia pyrina. Aderh.

1. Examine pear scab specimens and compare with apple scab in all details of symptoms, morphology and life history of the fungus, and control measures.

References.

(1) Duggar, B. M. Some important pear diseases. N. Y. (Cornell) Exp. Sta. Bul. 145: 616. 1898.

(2) Smith, R. E. Pear Scab. Calif. Exp. Sta. Bul. 163. 1905.

(3) Hesler & Whetzel. Manual of Fruit diseases, p. 332.

The life history of the pear scab organism is identical with that of the apple scab organism. There are slight morphological differences in the ascus stage of the fungus which may be observed under the microscope.

The important thing to be remembered is that the species of scab fungus which attacks apple cannot attack pear and vice versa, so that there need be no fear whatever that the disease will spread from apples to pears or from pears to apples.

CHAPTER XX.

Diseases caused by fungi - Basidiomycetes.

The basidiomycetes contain many of the most destructive and widespread plant diseases known. Here are classed such diseases as smuts and rust, which not only cause more loss to grains than any other cereal diseases but also attack a wide range of plants outside the grass family. Other important disease-producing basiodiomycetes are the bracket-and toadstool - forming, wood-rotting fungi, and Rhizoctonia of potato.

CORN SMUT*

Caused by Ustilago zeae (Beckm.) Ung.

This is probably the most serious disease affecting corn in the great corn belt of the central United States. As yet it is not serious in Oregon, but is met with occasionally in this state.

1. Examine specimens and describe symptoms. Note that the disease attacks all parts of the plant; ear, tassel, leaves, and stem, Draw each.

2. Mount some of the spores (chlamydospores) and draw. Find illustrations of germinating spores in books or bulletins. Note promycelium and sporidia.

3. Write up notes. Look up details of life history, distribution, losses, control, etc., in the references cited below.

References.

(1) Duggar, B. M. Fungous Diseases of Plants, p. 376.

(2) Stevens & Hall. Diseases of Economic Plants, p. 331.

(3) Arthur, J. C. & Stewart, W. Corn Smut. Indiana Exp. Sta. Report 12: 84 - 135. 1900.

(4) Hitchcock & Norton. Corn smut. Kan. Exp. Sta. Bul. 62: 169-212. 1896.

(5) Freeman, E. M. Stakman, E. C. The smuts of grain crops. Minn. Exp. Sta. Bul. 122: 37. 1911.

(6) Melchers, L. E. Smuts of grain and forage crops in Kansas. Kansas Exp. Sta. Bul. 210: 9. 1916.

*Note.- A general discussion of all smuts outlined in this manual will be found following the exercise on loose smut of barley.

7) Coulter, Barnes & Cowles. Text-Book of Bontany. Vol. I, p. 81.

8) Knowles, E. L. A study of the abnormal structures induced by Ustilago Zeae-mays. Jour. of Mycology. 5: 14. 1899.

Review.

When and where may infection take place in corn smut? Explain the germination f corn smut spores. How is the disease perpetuated? What part do promycelium and poridia play in the life history cycle? Discuss control measures.

OAT SMUT

Caused by Ustilago avenae (Pers.) Jens. and Ustilago levis (K. & S.) Mag.

The two species of Ustilago causing smut of oats affect the plants in a slightly different manner. One species, U. avenae causes what is known as "loose smut" because of the fact that the glumes are largely consumed by the fungus leaving the loose masses of smut spores almost entirely exposed. On the other hand, U. levis does not so nearly consume the glumes so that the masses of smut spores are more or less protected by the remains of the glumes. This form is known as covered smut of oats. It requires an oil immersion lens to distinguish any difference in the spores of the two species. With such high magnification the spores of the covered smut form are perfectly smooth while those of the loose form show minute spines or echinulations on the surface.

1. Symptoms.- Describe symptoms of both loose and covered smut forms. Draw.

2. Life history.- Mount spores and draw. If germinating spores showing the promycelium and sporidia are available, examine, and draw. If an oil immersion lens is available, try to distinguish the spores of the two species. Draw.

Infection takes place in the oat seedling and the mycelium of the fungus grows up inside the stem as the oat plant grows and finally smuts the panicle by destroying the ovaries and other flower parts.

3. Notes.- Give a detailed description of the two species of oat smut as to symptoms, life history, losses caused and control measures. Consult the following references:

References.

1) Duggar, B. M. Fungous diseases of plants, p. 372.

2) Jackson, H. S. Grain smuts. Ore. Exp. Sta. Crop Pest Report. 1911-12. p. 291-300.

(3) Swingle, W. T. Prevention of loose smut of oats. U. S. D. A. Farmers' Bul. 250: 13. 1906.

(4) McAlpine, D. The Smuts of Australia, pages 103-106.

Review.

Discuss oat smut as to symptoms, life history, and control measures.

STINKING SMUT OF WHEAT

Caused by Tilletia tritici (Bjerk.) Wint. and Tilletia levis Kuhn.

Stinking smut, sometimes known as "covered smut" or "bunt" is by far the most destructive form of wheat smut. Not only does it cut down the yield enormously in some cases, but reduces the value of any sound grain that may remain by the presence of the undesirable smut dust on and among the grains as well as by the offensive odor. Badly smutted grain is always docked heavily by the dealers.

1. Symptoms.- Compare smutted heads with healthy heads and describe the symptoms of smut. Remove some of the smut balls and break them open. Draw head and single smut ball. What part of the plant shows the smutted condition? Can you detect any difference in symptoms on plants attacked by the two species of Tilletia?

2. Life history of causal fungus.- Examine spores of the two species under the microscope. What is the difference. Draw both kinds. Look at illustrations of germinating spores on the chart. Note promycelium and sporidia. Compare with the same structures in oat smut and corn smut. What is the difference? Look up Kansas Exp. Sta. Bul. 210 and note the three groups or classes of smuts listed, pages 7-9. To which of these groups does stinking smut of wheat belong? To which group does oat smut belong? Corn smut? What are the distinguishable characteristics of each group? A discussion of these groups may also be found in McAlpine's Smuts of Australia, page 29, and in Ore. Exp. Sta. Crop Pest Report 1911-1912, p. 292.

References.

(1) Duggar, B. M. Fungous Diseases of Plants, p. 379.

(2) Jackson, H. S. Smuts of grain crops. Ore. Exp. Sta. Crop Pest Report 1911-1912. p. 291.

(3) McAlpine, D. Smuts of Australia, pages 70-85.

(4) Redd, Joseph. Treatment of stinking smut of Wheat. Colorado Exp. Sta. Bul. 79. 1903.

(5) Heald, F. D. Woolman, H. M. Bunt or stinking smut of wheat. Wash. Exp. Sta. Bul. 126. 1915.

(6) Heald, F. D. & George D. C. The wind dissemination of the spores of bunt or stinking smut of wheat. Wash. Exp. Sta. Bul. 151. 1918.

(7) Cardiff, Ira D. et. al. Report on fires occurring in separators in eastern Washington during the summer of 1914. Wash. Exp. Sta. Bul. 117. 1914. (For other references see lists under oat smut and corn smut).

(8) Barss, H. P. Grain smuts and their control. Oregon Experiment Station Third Crop Pest and Horticultural Report, 1915-20: 197. 1921.

(9) Heald, F. D. The stinking smut of wheat. Wash. Exp. Sta. popular Bul. 115. August, 1918.

(10) Heald, F. D. and L. J. Smith. The dusting of wheat for bunt or stinking smut. Wash. Exp. Sta. Bul. 168. June, 1922.

Review.

Describe the symptoms of stinking smut of wheat. Explain the life history of the smut fungus. What two species of fungus cause stinking smut? Distinguish between these two species. Is the life history of these two species different? Discuss control measures as related to life history. Is seed treatment entirely effective in all localities and under all conditions? Why? Discuss fully. Duscuss soil infection. What are "smut showers"? Discuss. Compare stinking smut with oat smut and with corn smut in all details.

LOOSE SMUT OF WHEAT

Caused by Ustilago tritici (Pers.) Rostr.

1. Examine specimens of loose smut of wheat and note symptoms. Compare with other smuts studied. Examine spores. Draw diseased plant and spores.

2. Look over references previously cited on smuts, especially McAlpine, Smuts of Australia, and compare the life history with that of stinking smut and oat smut. How does it differ? What does this difference have to do with control measures?

References

(Consult previous lists of references on smuts and also the following:

(1) Freeman, E. M. & Johnson, E. C. The loose smuts of barley and wheat. U. S. D. A. B. P. I. Bul. 152. 1909.

(2) McAlpine, D. The Smuts of Australia, p. 86.

Review.

Compare and contrast loose smut of wheat with stinking smut and oat smut in all details. Why can it not be controlled in the same manner?

COVERED SMUT OF BARLEY

Caused by Ustilago hordei. (Pers.) K. & S.

1. Describe the symptoms of this smut and compare with other smuts studied. Draw.

2. Compare the spores with spores of other smuts. Draw.

3. Covered smut of barley has the same life history as oat smut. What is that? How does that life history differ from that of the stinking smut fungus? To which of the three groups of smuts mentioned under stinking smut of wheat does this smut belong? If you have forgotten these groups look them up again in Kansas bulletin 210, pages 7-9, or McAlpine's Smuts of Australia, page 29, or Ore. Exp. Sta. Crop Pest Report 1911-12, page 292.

References.

(See any of the references previously cited on smuts.)

LOOSE SMUT OF BARLEY

Caused by Ustilago nuda (Jens.) K. & S.

1. Examine specimens and compare and contrast in all details with other smuts studied. How does this smut differ from covered smut of barley? From loose smut of wheat? To which of the three smut groups does this belong? Compare life history and control measures with those of all other smuts.

References

(Consult references previously listed for smuts).

(1) Barss, H. P. How to prevent grain smuts. Ore. Agr. College Ext. Bul. 292. 1918.

(2) Tisdale, W. H., J. W. Taylor and M. A. Griffiths. Experiments with hot water, formaldehyde, copper carbonate and chlorophol for the control of barley smuts. Phytopathology 13: 153. April, 1923.

Discussion of Smuts

The life history of the various smut groups is discussed by Freeman and Stakman (1) as follows:

"The smut parasite is a fungus plant composed of delicate, colorless threads, which live inside of the grain-plant, and which are so small that they can only be seen by the aid of the high power of the microscope. The fungus does not produce seeds, but reproduces itself by means of spores. The smutty mass, or smut-dust, is composed of these spores. Each spore is very small, so small that in some common smuts 5,000 of them placed side by side would make a line but an inch long. A smut-mass of corn, an inch in diameter, contains, therefore, many millions of s spores. The spores are single round cells, with thick walls, so that they are well protected against drying up or other injury. When the spores encounter favorable conditions they germinate and thus reproduce the disease.....

"The life of the smut.- It must first be noticed that all smuts are not alike. There are different kinds, and each smut has its own way of living. Moreover, the corn-smut does not cause smut in wheat, oats or barley. Neither does the oat-smut cause smut in wheat, barley or corn. Each smut is distinct. Smut-dust from corn can be mixed with wheat without producing any smut in the wheat. Furthermore, each smut has its own peculiar habits of life, and may differ from other smuts in many ways. For instance, corn-smuts get into any part of the corn plant, while stinking smut can get into the wheat-plant only when the wheat is very small; that is, when the seed is sprouting. On the other hand, two or more smuts may have similar habits of life, although each is a distinct disease. For instance, the stinking smut of wheat and the covered smut of barley both infect or get into the grain-plant (the one in wheat, the other in barley) in the sprouting stage, and both can be prevented by the same treatment; but the barley-smut cannot get into the wheat-plant and the stinking smut cannot get into the barley-plant.

"It is always well, in the treatment of any disease of plants, that the re. reasons for such treatment be known. The reasons for the treatment are almost always found in the life-story of the disease. It will be best, therefore, to recite the life-story of the common grain-smuts. As pointed out above, two or more smuts may have a similar life-story, although they may be different and distinct smuts, which cannot be transferred from one kind of grain to another.

"The common smuts of grains..... can be arranged in three groups, so r as the life-stories are concerned. The smuts of each group have in general a milar method of living. As a result, the treatments for the smuts in any one oup are quite similar. For instance, the life-stories of the oat-smut, the inking smut of wheat, the covered smut of barley, and grain-smut of sorghum, are l in general alike; and the treatments for their prevention are similar."..... eeman, E. M. and E. C. Stakman. The smuts of grain crops. Minn. Exp. Sta. Bul. 2. February, 1911.

We will designate the three groups of smuts mentioned above as follows:

Group I - Local infection group. - Corn smut.

Group II - Seedling infection group. - Stinking smut of wheat, covered smut of barley, oat smut, sorghum grain-smut.

Group III - Blossom infection group. - Loose smut of wheat, loose smut of barley.

Freeman and Stakman discuss these three groups further as follows:

I. "Life Story of Corn Smut.- The smut-mass of corn-smut varies greatly in ize, and may be found on any part of the plant. The smut-spores which come from his mass are blown out by the wind, and may of course be carried to a considerable istance; or the smut-mass itself may be broken off and thrown on the ground, or erhaps into a refuse or manure-pile. The spores in the manure-pile or on the round may start to grow. When they grow they send out small, delicate, colorless ubes; and these little tubes may form more spores; so that in a fresh manure-pile, he spores may actually increase during the winter. However, as the manure-pile rows old, the spores may die off, and hence old manure is better for fertilizing urposes than fresh manure. Having lived over the winter, either in the ground or n the manure pile, in early summer the smut renews its growth. Again its tiny pores are picked up by the wind and blown about. Some of these spores are blown pon a young corn-plant or on a young part of an older corn-plant. Such a spore ill germinate, producing a small tube, and this tube will grow into the corn-plant, enetrating the skin or epidermis. When the delicate little thread gets inside the orn-plant it continues to grow, both in length and by branching, and soon produces complex network of threads. These threads stimulate the corn-plant, so that it rows very fast in their neighborhood. All this time the threads are still growing nd feeding on the corn-plant. When this has gone on for some time, the smut-hreads commence to break up into small pieces, and each piece rounds up and ecomes a spore. The whole boil-like structure turns black and powdery, and we ave again the spore-mass or smut-mass.....

II. "Life-Story of Oat Smut, Stinking Smut of Wheat, Covered Smut of Barley and Sorghum Grain-Smut.- The life-stories of these four smuts, which are all common on Minnesota grains, are in all essential matters similar, so that they can be dis-cussed under one head. Of course there are slight differences even in the life-stories but these can be disregarded so far as this bulletin is concerned.

"In all cases, the spore-masses are found in the kernels of the grains. In
t-smut and the covered smut of barley, the chaff is also smutted to a certain
tent. When the smut spore-mass is broken, the spores get on the grain; as for
stance, in threshing of wheat and sorghum. A single grain of wheat or barley
y have clinging to it many hundreds or even thousands of these small spores.
; is in this stage that the smut passes the winter; that is, clinging to the
ed of the grain. When the seed with the spores on it is planted in the spring,
d the grain sprouts, the smut spores also commence to sprout by sending out
licate colorless threads. Some of these tiny threads grow into the grain sprout,
ercing the protecting leaves and penetrating to the center of the sprout.... It
s at this point that the so-called growing-point of the grain-plant is located.
e growing-point is the growing tissue which forms the leaves and new part of the
tem of the plant. The smut-threads live in this growing-point and grow up with
t, and continue to grow with it until the grain is ripe. The last thing that the
rowing-point produces is the head of the grain; and when this is still very young,
ven before it is out of the "boot", the smut-threads can be found in all parts of
t. As the head fills and becomes mature, the smut-threads increase; and finally,
t about harvest-time they break up into small pieces, each piece becomes a spore,
nd the spore-mass thus takes the place of the grain in a healthy plant.....

"As to the treatment of this class of smuts, it will be immediately seen that
he growing plant which already has the smut inside of it cannot be cured. It will
lso be immediately seen that the point of attack for treating is the seed, and all
hat is necessary to prevent a recurrence of these smuts, is to wash off or kill
he smut-spores that are on the seed before planting. If the spores are entirely
emoved, either by washing off or by killing with some solution, there will be no
mut in the following crop. (See Wash. Exp. Sta. Bul. 151.)

III. "Life- Story of Loose Smut of Wheat and Loose Smut of Barley.- These two
muts can be very readily distinguished from the other smuts on these two grains.
hey are sometimes called early smuts, since they discharge their spore-masses
arlier in the season. The spore-masses are looser than those of the second group
f smuts, and open up very early in the season. They commence to appear as soon
s the plants head out, and break as soon as, or even before, they get out of the
'boot". The wind blows them over the field. It will be noticed that the spores
re distributed at exactly the time when the grain-plant is in flower; that is,
t about heading-out time. Some of the spores are blown to the heads of healthy
lants, when the latter are in the flowering stage, and some are blown or are
ashed between the chaff into the flower. They get into the flower at about the
ime the seed commences to form, and they may lodge upon the outside of the young
eed-forming organ in the flower. Here the spore grows by sending out, as in
ther cases, a delicate tube, and this tube grows into the very young seed.....
Now, even in a very young seed, there is a little grain-plant, and the smut-threads
grow into this grain-plant. Here they remain while the seed grows in size and
fills out; and when this seed is ripe, although it looks like an ordinary healthy
seed, it has inside of its little plantlet the threads of the smut-plant. Such a
seed looks exactly like a healthy seed; and there is no way of detecting, from the
outside, any difference. It is well to compare such a seed with a seed on which
spores of the preceding group of smuts are clinging. In the case, for instance,
of stinking smut of wheat, we would have a wheat-seed with spores on the outside;
while in the loose smut of wheat we have a wheat-seed with the smut-plant inside
of the little plant in the seed. It will be seen at a glance that the loose smut,

ith its smut-plant inside of the seed, is more difficult to treat than the seed ith the smut spores on the outside. This is the reason why ordinary treatment, uch as is recommended in the preceding group of smuts, has no effect on the oose smut of barley or on the loose smut of wheat, because such treatment does ot reach inside of the infected seed. Now, when the infected seed, with its mut-plant inside, is sown in the spring-time, it sprouts just as an ordinary ealthy seed. As the young plant in the seed begins to grow, the smut-threads nside of this plant also commence to grow, and they keep pace with the growth of he grain-plant. From this point on, the behavior of this smut is like the be- avior of the smuts of the preceding group; that is, the smut-threads live in the rowing-point of the grain-plant and remain there until the head is formed. Then he smut-threads grow on through the young kernels and chaff of the new head, and inally break up into small pieces, which are rounded up into little spores, and ve have again the smut-mass of the field.

"As to the treatment, since an infected seed has no spores on the outside, ut has the smut inside of the seed, some methods have to be devised other than those used for smut of wheat and barley. Such are described below. They are, briefly, built upon this principle: The seed is soaked in cold water for several hours. In its water-soaked condition, heat can be carried through the seed more uniformly and quickly than when it is dry. The soaked seed is then treated in hot water, and then the smut-plant inside of the seed is killed; while the seed is not at all, or only slightly, injured by the treatment.....

"Since the treatment for one group of smuts may have no effect on the other, it is absolutely necessary for a farmer to know which smut he is dealing with. It then becomes an easy matter to apply the proper remedy. For instance, in treating smut in barley, a farmer who treats his barley with the formalin method will get rid of the covered smut of barley, but will not get rid of the loose smut of barley. The same holds true of wheat."- Freeman, E. M. and Stakman, E. C. The Smuts of Grain Crops, University of Minnesota Agricultural Experiment Station. Bulletin No. 122: 37-45. 1911. (See Jour. Agr. Res. 19: 263).

Control.

The following discussion of control measures is quoted from Barss (2).

Hot Water Treatment.
(For loose smuts of wheat and barley)

(1) "Soak grain to be treated 4 to 6 hours in cold water.
(2) "Taking a convenient amount of grain in a screen-lined basket or other container, plunge for one minute into a barrel of water kept at a temperature close to 120° Fahrenheit to warm the seed up.
(3) "Remove and plunge at once into another barrel where the water is kept at exactly 129° Fahrenheit for wheat and 126° Fahrenheit for barley. Keep wheat immersed for 10 minutes and barley for 13 minutes..
(4) "Remove exactly on time and plunge into cold water to stop the action of the heat.
(5) "Spread out in a thin layer to dry. If not to be planted at once, dry the seed out completely before sacking up again.

"The important things to bear in mind are to keep the temperature constant and correct and to be exact in the time of treatment. A floating dairy thermometer should be used and more boiling water added from time to time as may be necessary, but the water must be thoroughly stirred so as to get a uniform temperature through out. Too high a temperature or too long treatment in the hot bath will injure the grain, while too low a temperature or too short exposure will not kill the smut.....

"Resistant Varieties.- Mr. H. M. Woolman, Cooperating Field Assistant of the United States Bureau of Plant Industry, with headquarters at Corvallis has within a period of two years tested out over a thousand strains and varieties of wheat for bunt resistance in plantings at Corvallis, Astoria, Moro, Hermiston, Union, and Medford. Several very promising resistant strains have appeared. Among these may be mentioned some of the Turkey selections made at the Moro Branch Station by Mr. D. E. Stephens. Several other highly resistant varieties have been found, among them one which thus far appears to be totally immune. Using these as parent stocks to furnish the element of resistance there is every reason to expect that progeny may be developed having also the highest commercial value. The final perfecting of well adapted, smut-resistant grain varieties would be a tremendous, permanent advantage to Oregon, abolishing the need for chemical treatments and forever annihilating the otherswise apparently insuperable soil-infection problem...

"Formaldehyde Method.- This is the common method in use for oats and may also be used for wheat and barley when soil and temperature conditions are favorable for prompt germination.

Formaldehyde Formula

"1 pint formaldehyde (37% to 40% strength) to 40 gal. water (or 1 ounce--two tablespoons--to 10 quarts water).

"Wet all the grain thoroughly either by sprinkling at the rate of one gallon of solution per bushel, or by pouring loose into the solution, or by soaking partly-filled and loosely-tied gunny-sacks for five minutes or so until thoroughly wet throughout, or run through a treating machine. Loose grain should be piled up after being treated and covered with canvas or sacks for two hours. Sacked grain should be drained and allowed to stand two hours. Then plant at once or spread out to dry and plant within a few hours.

"Unless wheat is absolutely free from smut balls, it should be poured into the solution, thoroughly stirred, and the smut balls skimmed off, as the solution will not disinfect solid smut balls and any such on passing through the drill will break and recontaminate the seed as it runs out.

"Dry Formaldehyde Method for Oats.- This is a new and promising dry method of treating, which has given successful results wherever used on oats. Wheat may be injured by this method. It has not been tested in Oregon on barley.

"Use 1 part of formaldehyde (37% to 40%) to 1 part of water.

"Spread the grain on a clean floor. As the grain is shoveled from one pile o another, each shovelful is sprayed with a small quart hand sprayer (atomizer), eld close to the grain, using 1 quart of solution to 50 bushels of oats.

"When all are treated, pile in a heap, cover for four or five hours to retain umes, then uncover, air out, and plant at once or in a few hours.

"Bluestone-Lime Method.- Bluestone (blue vitriol) is often injurious to oats. It is, however, the best method for wheat and barley where soil contamination is feared or where the soil and temperature conditions are not favorable for prompt germination. The coating of bluestone remaining on the grain serves to disinfect the soil slightly around the grain - although it will by no means eliminate soil infection entirely.

"Bluestone when used alone is often very injurious to the vitality of wheat. This is partly due to the large percentage of kernels (often 80% or 90%) which have the skin broken over the germ by ordinary threshing methods. In order to neutralize the bluestone and thus prevent a large part of this injury it is necessary to dip the treated grain in a lime-water bath.

Bluestone Formula

"1 pound bluestone (copper sulfate) to 5 gallons of water. (To this formula 1 pound of salt is sometimes added).

"Suspend the chemicals in gunny sacking at top of the required amount of water in tank or barrel until dissolved completely, and stir the solution thoroughly before beginning the treatment, so as to get the solution uniform. Or to hasten the solution, the chemicals may first be dissolved in hot water. Do not use bluestone solution in metal containers, excepting copper, as it will corrode them.....

"Pour the grain slowly into the solution, stir thoroughly and skim off all smut balls. If the grain is absolutely free from smut balls it may be dipped into the solution in half- or third-filled gunny-sacks, which should be soused up and down to insure complete wetting of all the grain. Allow at least three to five minutes for the soaking, then drain for about fifteen minutes.

Lime Bath Formula

"1 pound of quick lime to every 10 gallons of water. (Slake required amount of lime in usual manner and dilute according to formula).

"Then dip or pour the grain into the lime bath. The lime solution should be renewed whenever it loses strength, which can be determined by the blue color or by the fact that it will turn blue litmus paper red. Agitate the grain and allow the lime to act for at least three to five minutes. Remove the grain, drain, allow it to dry, and plant as soon as possible. If it cannot be planted promptly, it must be spread out and dried rapidly and completely before setting away."- Barss, H. P. Grain Smuts and Their Control. Third Crop Pest and Horticultural Report, Oregon Agricultural College. 1915-1920: 198-202.

Copper Carbonate Dust.

A new and quite promising method of dusting seed wheat with copper carbonate for the control of bunt has recently come into use. It is yet in the experimental stage but great success has been secured with it in some cases. Some of the more interesting facts concerning the copper carbonate treatment are discussed in the following extracts from Washington Experiment Station Bulletin 168 (3).

"Historical Statement Concerning the Use of Copper Carbonate.- Copper carbonate as a fungicide for seed disinfection has been disregarded as its extreme insolubility in water was thought to exclude the possibility of effective action. In the form of a finely powdered dust it was first tested by Darnell-Smith in New South Wales as a fungicide in the control of wheat smut. On the basis of tests conducted in 1915 and 1916 he reported in 1917 that copper carbonate dust gave better results than either formaldehyde or bluestone. A more complete report was issued in 1919, with the claim that machine-treated grain was free from smut while hand-treated grain showed a slight amount of smut. The copper carbonate dust treatment caused no injury to seed or to young plants and also resulted in very substantial increases in yield. The results of Darnell-Smith were questioned by Perkins in 1919 who stated that 'we can not, for instance, imagine that a substanc wholly insoluble in water can have high fungicide properties'. A discussion of this criticism was published by Darnell-Smith in 1921.

"The excellent results obtained in Australia soon attracted the attention of American investigators. Mackie and Briggs first reported experiments in Californi which corroborated the reports from Australia. They reported in 1921 that spring wheat treated with 2 ounces of copper carbonate dust per bushel was free from smut and suffered no reduction in germination, while the untreated check gave 6.2 per cent of smutted heads.

"Lambert and Bailey have just recently reported perfect control of both bunt of wheat and smut of oats by using 2 ounces of copper carbonate dust per bushel. No reduction in germination of oats resulted from using 2 ounces per bushel, while 2 to 10 ounces of the copper carbonate caused no appreciable injury to wheat.....

"Formula and Methods of Application of Copper Carbonate Dust.- On the basis of completed trials our present recommendation is the use of 2 ounces of finely powdered copper carbonate, testing 50 per cent or more of metallic copper, per bushel of wheat. The powder should be fine enough to pass through at least a 100-mesh sieve. The effectiveness of the treatment depends on the uniform distribution of the finely divided particles over the surface of the grain. Two ounces per bushel gives a complete coating, and no added protection has been afforded by using 4 ounces per bushel. Work now in progress will tell whether it will be possible to use less dust per bushel and still obtain effective protection.

"The application of the copper carbonate dust may be made in a number of different ways. In our earliest experimental work the treatment was carried out by shaking the seed and the requisite amount of copper carbonate in a closed container. A barrel churn has been used with good results, while in some larger operations a small concrete mixer has been tried. In some of the demonstration trials the dusting has been accomplished by emptying the wheat and the desired amount of copper carbonate on a canvas and shoveling over the mixture until the

grain became thoroughly coated, but this last method is not recommended as it is felt that it gives the least affective distribution of the dust and offers more opportunity for persons doing the treating to inhale the irritating and poisonous dust particles. A special dusting machine has been designed which is effective and also convenient, and it is expected that this or a similar machine will come into general use.....

"Advantages of the Copper Carbonate Treatment.- The advantages of the dust treatment are so pronounced that this method of control may be expected to replace the older wet treatments with bluestone or formaldehyde. It is our prophecy that we are now beginning the 'copper carbonate dust period of seed disinfection'. The important advantages of this copper carbonate dust may be briefly enumerated as follows:

1. The elimination of the inconvenient and disagreeable soaking methods.
2. The convenience of being able to treat seed days or even months before it is needed for use, thus making possible the utilization of spare time.
3. The elimination of seed injury and retarded growth, with even an improvement in germination over that of untreated seed. This will make possible a reduction in the amount of seed used per acre and should result in a considerable annual saving as soon as the method comes to be generally practiced.
4. The elimination of danger from seeding in the dry, making it possible to seed in the 'dust', rather than being obliged to wait for rain.
5. Probable increased yields due to better stands, no retarding influence of the fungicide, and better control of smut.
6. A probable increased protection from reinfection of treated seed by smutty sacks, broken smut balls or from the wind-borne spores that are generally present in our summer fallow fields."- Heald, F. D. and Smith, L. J. The Dusting of Wheat for Bunt or Stinking Smut. State College of Washington Agricultural Experiment Station Bulletin No. 168: 5-14. 1922.

Cases where seed treatment is not always completely successful.

The conditions in the great wheat growing sections of Oregon and Washington east of the Cascade Mountains are different from the conditions in most other wheat growing sections of the United States. This difference makes it impossible to completely control the bunt or stinking smut in this region by the ordinary seed treatments that are successful in other parts of the country.

Washington Experiment Station bulletin 151 (4) and popular bulletin 115 (5) explain these conditions and suggest means of over-coming the difficulty to some extent. The summaries from these bulletins are quoted below.

The Wind Dissemination of the Spores of Bunt or Stinking Smut of Wheat.

"Summary.- Seed treatment, the effective practice for the control of bunt or tinking smut of wheat in other wheat-growing sections, has not been effective for inter wheat in the Palouse country.

"Bunt is one of the cereal smuts in which the infection takes place only hrough young seedlings, either from seed-borne spores or from spores already in he soil.

"The possibility of a wind-dissemination of the spores of bunt has been ecognized. It has been shown that wind-borne secondary spores or sporidia play o part in the spread of smut, since there is no spread of smut from an infected o an uninfected field during a single season. Scattering of spores from smutted eads previous to harvest is of minor consequence, but during the threshing operaions the smut balls are broken up and much of the smut dust is carried out through he stacker. Much of this smut must ultimately come to rest on the surrounding ields.

"The wide-spread wind-dissemination of the spores of bunt was suggested by he visible clouds of smut dust which issue from threshing machines whenever the un is in a smutty field; the presence of smut spores on vegetation distant from ny threshing operations; the production of smutty wheat when clean treated seed as sown on land, which had not previously produced a crop of smutty wheat; and lso by the general ineffectiveness of seed treatment for fall-sown wheat.

"Positive evidence of this widespread dissemination of the spores of bunt as been obtained by the microscopic analyses of the washings of foliage taken rom stations distant from any threshing operations. This was further substantiated by the use of water spore-traps, which have made possible the determination of the actual number of spores falling on a given area.

"During the threshing season there is a 'smut shower' or spore-fall in the icinity of Pullman, which begins in August, increases to a maximum and gradually ubsides. During the season of 1916 the maximum fall occurred from the 5th to the lth of September. The exact time of the maximum will depend in part on the ocation, the period when threshing operations are the most numerous, and also pon climatic factors.

"The dry conditions which prevail during the threshing period, together with requent winds of high velocity, and a farming system which includes summer fallow nd winter wheat, have combined to make wheat smut especially severe in the Palouse ountry.

"The greater amount of winter wheat is seeded in summer fallow which has reeived countless numbers of wind-blown spores; the maximum amount of seeding enerally follows the period of maximum spore-fall, and at this time both temperaure and moisture conditions are especially favorable for smut."- Heald, F. D. nd George, D. C. The Wind Dissemination of the Spores of Bunt or Stinking Smut f Wheat. State College of Washington Agricultural Experiment Station. Bulletin o. 151: 22-23. 1918.

1. "Summary.- Wheat smut, the most important plant disease of the Pacific Northwest, takes an enormous annual toll.
2. "The control of wheat smut in the Inland Empire presents a problem different from that which confronts farmers in other parts of the United States.
3. "Seed treatment, the effective practice in other wheat growing sections, must be supplemented by other practices.
4. "Under present Palouse conditions it is not possible to produce absolutely smut-free wheat, since many wind-blown spores reach the uninfected fields.
5. "Seed treatment alone is not effective since there is a general and widespread dissemination of smut spores during the threshing season, so that all summer fallow fields are heavily seeded by the regular planting time.
6. "The 'smut shower' or spore fall increases during the threshing season to a maximum and gradually subsides.
7. "Seed treatment is generally effective for spring wheat, since wind-blown smut does not survive the winter.
8. "Experimental seedings have shown that either early or late plantings are either free from smut or show a low percentage of infection.
9. "Beginning with seedings early in August there is a gradual increase in the per cent of infection, the maximum being reached about two weeks after the maximum spore fall; following the maximum there is a gradual reduction in the per cent of infection to the end of the growing season.
10. "Smut exhaust fans offer much of promise as a means of preventing fires in separators, improving the quality of the wheat, and in reducing the amount of wind-blown smut."- Heald, F. D. The Stinking Smut of Wheat. ashington Agricultural Experiment Station popular Bulletin No. 115: 14. 1918.

General Review on Grain Smuts.

Be able to identify every grain smut studied and to give the exact life istory of the causal fungus. Make out a list of all smuts studied and classify nem in the three infection groups. Discuss control measures for each of the nree groups. Describe the hot water method; for which smuts is it recommended? ny? Describe the formalin dip method. What are its advantages and disadvantages? or which smuts recommended? Describe the formalin sprinkling method. For which muts recommended? Describe the copper sulfate method. For which smuts recomended? What are the dangers to be avoided with each method of treatment? Under nat conditions is seed treatment, even though properly executed, not effective n controlling smut? How may these other difficulties be overcome? Based on vailable statistics, how much damage are grain smuts responsible for in the nited States? Describe the dry copper carbonate seed treatment for smut. iscuss the problem of smut immunity in grains.

References.

1) Freeman, E. M. and E. C. Stakman. The smuts of grain crops. Minn. Exp. Sta. Bul. 122. 1911.

2) Barss, H. P. Grain smuts and their control. Ore. Agric. Exp. Sta. Crop Pest and Horticultural Report 1915-1920: 198-202. 1921.

) Heald, F. D. and L. J. Smith. The dusting of wheat for bunt or stinking smut. Wash. Exp. Sta. Bul. 168: 5-14. 1922.

) Heald, F. D. and D. C. George. The wind dissemination of the spores of bunt or stinking smut of wheat. Wash. Exp. Sta. Bul. 151: 22-23. 1918.

) Heald, F. D. The stinking smut of wheat. Wash. Exp. Sta. Popular bul. 115: 14. 1918.

ONION SMUT

Caused by Urocystis cepulae. Frost.

In certain onion growing sections onion smut is an important factor in cutting wn yields. Due to the fact that the onion smut spores remain alive in the soil r such a long period of time, it is sometimes necessary to abandon onion raising badly infested fields for a great many years. The spores are said to remain able in the soil for ten or twelve years.

1. Examine specimens and describe the symptoms. Draw.

2. Examine sections of diseased plant or mount some loose spores. Do the ores look like other smut spores studied? What is the difference, if any? Draw.

References

) Duggar, B. M. Fungous Diseases of Plants, p. 381.

) Selby, A. D. Onion Smut. Ohio, Exp. Sta. Bul. 122. 1900.

) Sirrine, F. A. & Stewart, F. C. Experiments on the sulfur-lime treatment for onion smut. N. Y. (Geneva) Exp. Sta. Bul. 182. 1900.

) H. S. Jackson. Onion smut, Ore. Exp. Sta. Crop Pest Report 1913-14. p. 267.

) Barss, H. P. Onion smut control. Oregon Experiment Station. Third Crop Pest and Horticultural Report 1915-1920: 187. 1921.

) Anderson, P. J. Development and pathogenesis of the onion smut fungus. Mass. Exp. Sta. Technical Bul. Nov. 4. 1921.

GEREAL RUSTS

There are seven species of rust which attack the various kinds of grain crops. These are: Black Stem Rust of wheat, barley, oats, rye and other grasses; Leaf Rust of wheat; Stripe Rust of wheat and various grasses; Leaf Rust of rye; Leaf Rust of barley; Leaf or Crown Rust of oats; and Corn Rust.

Review the life history of the Black Stem Rust which you studied in general botany, then note the following grouping of the above rusts under their respective cereal hosts with the corresponding alternate host where known. Note that there are different strains or biologic forms of the stem rust for the different hosts, wheat, rye and oats.

Cereal host and name of rust.	Alternate host.
WHEAT.	
Stem rust caused by *Puccinia graminis tritici*.	Common barberry (*Berberis vulgaris*)
Leaf rust caused by *Puccinia triticina*.	Meadow-rue (*Thalictrum* spp.)
Stripe rust caused by *Puccinia glumarum*.	Unknown.
BARLEY.	
Stem rust caused by *Puccinia graminis tritici*.	Common barberry (*Berberis vulgaris*)
Stem rust caused by *Puccinia graminis secalis*.	" " " "
Leaf rust caused by *Puccinia simplex*.	Star of Bethlehem (*Ornithogalum*)
Stripe rust caused by *Puccinia glumarum*.	Unknown.
RYE.	
Stem rust caused by *Puccinia graminis secalis*.	Common barberry (*Berberis vulgaris*)
Leaf rust caused by *Puccinia dispersa*.	*Anchusa* spp.
Stripe rust caused by *Puccinia glumarum*.	Unknown.

ATS.
Stem rust caused by
Puccinia graminis avenae. - - - - - - - - - Common barberry

Leaf rust caused by
Puccinia coronata. - - - - - - - - - - - - Buckthorn (Rhamnus cathartica)

CRN.
Rust caused by
Puccinia sorghi. - - - - - - - - - - - - - Wood-sorrel (Oxalis sp.)

STEM RUST OF GRAINS AND OTHER GRASSES

Caused by Puccinia graminis. Pers.

This species of rust has been investigated and experimented with perhaps more than any other species of rust. It was with this rust that the research work was conducted which first made known the fact of heteroecism (having two hosts) in rusts.

This rust has five types of spores in its complete life history, namely, pycnospores (spermatia), aeciospores (aecidiospores), uredinio-spores (uredospores), teliospores (teleutospores) and sporidia (basidiospores). The first two of these spore forms occur on the common barberry (Berberis vulgaris), the third and fourth forms occur on grains and grasses, and the fifth form occurs saprophytically on germinating teliospores which have come through the winter in viable condition. (For a discussion of these spore forms see Duggar, B. M., Fungous Diseases of Plants, page 385, or McAlpine, Rusts of Australia, pages 7-28).

1. Symptoms.

a. On barberry leaves.- Observe the pustules (pycnia or spermogonia) which bear the pycnospores. These are located on the upper side of the leaf. They may be seen to better advantage with the aid of a hand lens, a dissecting microscope or a binocular microscope. On the under side of the leaf find the clusters of cups (cluster-cups, aecia, aecidia). Make a habit sketch of the leaf to show each kind of fruiting body.

b. On grain plants.- (1) Find the pustules (uredinia) which bear the red spores (urediniospores or uredospores.) This is usually known as the red-rust stage or summer stage of the rust. Draw. (2) On the same plant or another plant find the pustules (telia) which bear the black spores (teliospores or teleutospores). Draw.

2. Morphology and life history of the stem rust fungus.

a. The aecial stage.- Examine, under the microscope, sections of barberry leaf cut through the pycnia and aecia. Draw a section to show relation of fungus to host tissue. Show the chains of aeciospores in the cup.

b. Uredinial stage.- Mount spores of the red rust stage from wheat or her grain and draw under high power lens of the microscope.

c. Telial stage.- Mount teliospores and draw.

d. Examine shriveled grain from a badly rusted field.

3. Notes.- Write a complete description of the stem rust in all stages. scribe the complete life history cycle. Look at illustrations of germinating liospores and try to understand the part which the promycelium and sporidia play the life history of the fungus. Make a diagram to illustrate the life history rcle.

Discussion

Before taking up a special discussion of the stem rust, a few important facts oncerning the rusts in general should be mentioned. These points are brought out y Duggar (1) in the following brief account:

"Heteroecism.- In this order of fungi there has been developed not only great iversity in form and character of spores, and in the relationships of these types ne to another, but also a very definite relationship between the different spore ypes and the host plants. Where more than a single spore type is present a pecies may either complete its entire life cycle on one host, or it may require wo plants for complete development (in a few cases three) in regular order. The ormer group of rust fungi are termed autoecious and the latter heteroecious. utoecism is the rule among fungi generally. Heteroecism is better developed in usts than in any other group of living organisms, and it is with one or two exceptions confined to the rusts, so far as the fungi are concerned. There are more han 150 cases of heteroecism which have been experimentally demonstrated in this rder, and this number will be greatly increased as the experimental work proceeds. pon such hosts as the grasses, sedges, rushes (Gramineae, Cyperaceae, Juncaceae), nd allied plants, the spore forms produced are quite generally the uredo and eleuto stages, or one or the other of these; and in general, so far as the experiental work has been carried, such fungi have other stages, at least an aecidial tage, upon some dicotyledonous host. Indeed, in only one group of cases (the pecies of Puccinia on Phalaris) is the aecidial stage produced on another monootyledonous host. Again, in no case of heteroecism has the aecidiospore been ound to be capable of infecting also the host upon which it is borne. Since the eleutospore germinates by the development of a promycelium and sporidia, and in o other manner, it is precluded that the teleutospore may infect directly the host pon which it is produced. The uredospores alone possess this capacity.

"In general, it would seem that the period of incubation may vary from eight o twenty days during the growing season, for most of the species of rusts. The ime, however, will vary in the same species under different climatic conditions.

"The terminology of spore combinations.- Based upon the association of spore orms, that is, upon the number and kind of spores present in a particular species, chroeter has proposed certain very convenient type names as below. First, however, t should be stated that the spermogonial, aecidial, uredo, and teleuto stages are

respectively represented by O, I, II, III, and it is here unnecessary to consider the fifth or sporidial stage; the types then, are as follows:

"Eu forms with all stages; or O, I, II, III present.
Brachy forms with aecidia omitted; or O, II, III present.
Opsis forms with uredo omitted; or O, I, III present.
Hemi forms with spermogonia and aecidia omitted; or II, III present.
Micro forms with teleutospores only; or III present, germinating only after a resting period.
Lepto forms with teleutospores only; or III present, germinating immediately.

"It is interesting to note that in the far North or in Alpine regions, as Fischer shows, the micro and lepto forms predominate. Aecidia occur alone in considerable number, and also hemi forms. Many hemi forms, particularly in such genera as Uromyces and Puccinia, have been insufficiently investigated, and will doubtless prove to be eu forms, mostly heteroecious, that is, euhetero forms."- Duggar, B. M. Fungous Diseases of Plants. 1909: 387-388.

Stem Rust

The following data concerning the black stem rust is quoted from Stakman (8) in the Dep't of Agriculture Yearbook for 1918.

"Damage done by black stem rust.- The black stem rust is found practically wherever grain is grown in the United States. It is also found generally in Canada, South America, Europe, Asia, Africa, and Australia. In many sections of the United States black stem rust is the limiting factor in grain production. While it is especially destructive to wheat, it does a great deal of damage also to oats, barley and rye. It is most serious on spring wheat, but sometimes it also may cause enormous losses of winter wheat. In the Gulf States it is sometimes so serious as to make it entirely unprofitable to grow wheat and other small grains.

"At irregular intervals rust develops ravaging epidemics which sweep across great areas of the grain-growing regions and almost completely destroy immense quantities of grain. Some rust epidemics have been so serious and widespread as to cause a decided shortage of food-stuffs. It is necessary only to recall the terrible epidemics of 1904 and 1916. It was estimated that in 1904 the rust caused a loss of $20,000,000 in Minnesota, and the two Dakotas. In 1916 the production of wheat in Minnesota, North Dakota, South Dakota, and Montana was reduced by over 200,000,000 bushels from that of the previous year. This appalling loss was caused very largely by the black stem rust. After making due allowance for the reduction in acreage in some of these States and after making allowance for somewhat unfavorable weather, the fact remains that the principal cause of this enormous loss was the black stem rust. Thousands of acres of wheat never were cut. The grain would not have paid for harvesting and thrashing. Hundreds of farmers in the spring-wheat region were practically ruined on account of the almost complete failure of the wheat crop. In addition to the enormous reduction in yield, the quality of the wheat produced was very inferior. Much of it weighed only 40 to 50 pounds to the bushel.

"In 1916 the average yield of spring wheat in North Dakota was only 5½ bushels per acre, compared with an average yield of more than 18 bushels an acre. In South Dakota the average yield in 1916 was not quite 7 bushels per acre, as compared with 17 bushels in 1915 and a 10-year average of 10½ bushels. The greatest reduction in total production occurred in North Dakota. The production in that State in 1915 was about 150,000,000 bushels while in the severe rust year of 1916 it was less than 40,000,000 bushels. The production in Minnesota in 1915 was about 70,000,000 bushels, but it dropped to 28,000,000 bushels in 1916. The most conservative estimate places the loss of wheat in the United States due to the black stem rust in 1916 at 180,000,000 bushels, while the loss in Canada was estimated at about 100,000,000 bushels. In Canada and the United States, therefore, the black stem rust destroyed at least 280,000,000 bushels of wheat in a single year. To this must be added the loss of oats, barley, and rye. Reports showed very clearly that the loss of barley and oats in some localities often amounted to as much as 15 to 25 per cent of the crop.....

"Grains and Grasses Attacked by Black Stem Rust.- Wheat, oats, barley, rye, spelt, emmer, einkorn, timothy, redtop (Agrostis alba), orchard grass (Dactylis glomerata), and various other forage grasses are attacked by the black stem rust. In addition to the cereals and cultivated grasses many wild grasses also are subject to rust. Among the most important of these grasses are wild barley or squirrel-tail grass (Hordeum jubatum), quackgrass (Agropyron repens), slender wheat-grass (Agropyron smithii), awned wheat-grass (Agropyron caninum), bottle brush-grass (Hystrix patula), practically all of the wild rye-grasses (Elymus spp.), fescue grasses (Festuca spp.), koeleria (Koeleria cristata), sweet vernal grass (Anthoxanthum puelli), and several brome-grasses (Bromus spp.).

"Although all of these plants can be attacked by the black stem rust, there are forms or races of this rust which act somewhat differently. For instance, there is one race of black stem rust on wheat and barley. This race does not attack oats or rye normally. There is also a race on rye and barley which does not attack wheat and oats. Again, the race on oats attacks only oats and certain grasses. The race on timothy attacks only timothy and several wild grasses. All of the races can attack several of the wild grasses, but not all of them can attack the same grasses. This explains apparently conflicting observations. For instance, it is quite possible that a field of oats might be badly rusted while a near-by wheat field might be almost entirely free. In the same way, a wheat field might be badly rusted and a neighboring field of rye might be practically free from rust, because the forms of rust on these different crops are different.....

"How does rust live over winter?- There has been a popular belief that the rust lives over winter in the seed or in the red stage, as well as in the black stage. Considerable work has been done to determine just how the rust lives through the winter.....

"Does the Red Stage Live over Winter?- For the past two years the Department of Agriculture has been studying the question of the overwintering of the red-spore stage. It is well known that the leaf rusts overwinter in the red-spore stage. It has been shown clearly that the black stem rust does not overwinter commonly in the red-spore stage except in the extreme South and on the Pacific coast, where the rust can continue to develop on fall-sown grains practically throughout the winter. Thus, in the Gulf States and in California the red stage of rust can continue to develop practically the year round. However, it is

erfectly clear that the rust does not overwinter commonly in the red-spore stage xcept in the extreme South, on the Pacific coast, and in some protected mountain alleys. Experiments in the winter of 1917-18 prove conclusively that the red tage did not live through the winter as far south as Jackson, Tenn. It was shown lso that the rust did not overwinter in the red-spore stage in Oklahoma, Kansas, issouri, Kentucky, Nebraska, or in any of the States farther north. All the vidence available at present shows that if the red stage of rust does live through he winter at all in the Northern States, it does this so rarely as to be of little mportance in starting rust epidemics.

"The question naturally arises whether the rust spores which overwinter in he south could not be blown northward in the spring and infect the growing grain. n this way the rust might travel from south to north by successive stages as the rop develops. Evidence based on careful observations shows quite clearly that his does not occur. The rust develops on barberry plants in the North and spreads o grains and grasses quite as early in the spring as the rust begins to become eneral in the South. Then, too, the form or race of wheat rust which is common n the South can not cause rust on hard spring wheats or on most of the hard winter heats of the North. Even if the rust did blow up from the South, therefore, it ould do very little damage in the North.

"The overwintering of the Black Spores.- In the Gulf States the weather is ild enough to enable the red spores to live through the winter, but in the upper issisippi Valley only the black spores live through the winter to any extent. n the spring they germinate and infect the barberry. The rust spreads from the arberry to grains and grasses and continues to spread as long as weather condi- ions are favorable. Field observations show clearly that in the northern half of he Mississippi Valley the barberry gives the rust its start in the spring.

"Conditions which favor rust development.- Weather affects the development of ust in several ways. It is plain that if rust is to develop and spread, the red r summer spores must be blown from plant to plant. Strong winds carry the spores ong distances and therefore enable the rust to develop over a wide area. But ven though the spores have scattered widely they will not germinate unless the onditions of moisture and temperature are favorable. Heavy dews and fogs or teady, quiet rains furnish the best conditions for spore germination and conse- uently for rust infection. Heavy driving rains are probably not so favorable for nfection, because they wash many of the spores from the plants on which they have allen.

"After the rust parasite has entered a plant it will develop most rapidly hen the weather is rather hot and muggy. At low temperatures the rust develops uch more slowly, and it may also be checked by hot, dry weather. The weather lso may be favorable for infection but not for rust development after infection. r it may be favorable for the growth of rust at one time during the season and ot at another. When the right conditions occur at just the right times epidemics evelop.

"The variety of grain grown and the condition of the plants affect the apidity of the growth of the rust parasite. Soil conditions influence rust evelopment in so far as they affect local conditions of moisture and temperature nd the growth of the grain plants.

"How to reduce rust losses by cultural methods.- Rust can not be prevented ntirely, but the losses which it causes can be reduced. Proper soil management, rly sowing, the use of early-maturing and resistant varieties, the destruction weed grasses which can be attacked by rust, and the eradication of the common rberry all will aid in reducing rust losses.....

"Resistant varieties of grain.- There is some hope of reducing rust losses by he use of rust-resistant varieties. More is known about the resistance of wheat arieties than about that of any other kind of grain. It has been known for many ears that some varieties of wheat do not rust as heavily as other varieties under he same conditions. The durum wheats, as a group, have been considered fairly esistant to stem rust. Not all varieties of durum wheat are resistant, but a reat many of them are. This makes it possible to grow fairly good crops of some urum wheats when the bread wheats are injured severely by the rust.

"Until recently no good bread wheat was known which was resistant to rust. selection from the Crimean group made at the Kansas Agricultural Experiment tation several years ago, however, was tested for rust resistance and was found o be almost entirely immune under Kansas conditions. It seemed, therefore, that he rust problem in the winter-wheat regions might be near solution. However, it as since been found that there are many races or forms of wheat rust. They differ rom each other chiefly in their ability to attack different varieties of wheat. ome of the varieties of durum wheat which are quite resistant to rust forms in any localities are quite susceptible to the forms of rust occurring in other ocalities. In the same way, some varieties of hard spring wheat which rust most eavily in the spring-wheat region do not rust severely when grown in the South nd were almost entirely free from rust, while the native soft winter wheats in hat region were rusted heavily. In the same way, the resistant Kansas variety, anred, was very resistant when grown in Kansas but rusted in some other parts of he United States.

"No one variety now known is resistant to all the forms of rust which occur n the United States. No one rust-resistant variety of wheat, therefore, can be ecommended for universal use. The problem is a local or regional one, and arieties should be selected with this in mind.....

"It is perfectly clear, however, that rust resistance alone should not commend variety of wheat for general use. It must combine other desirable characters ith its rust-resistant quality. For this reason much crossing and selecting have een done to combine the rust-resistant character of some varieties with the high-yielding and good thrashing and milling qualities of other varieties. Some success as been attained, but not enough progress has been made to give any hope that the ust problem may be solved entirely in this way in the near future. Until it is known how many races of the stem rust there are, where they occur, and which varie-ties they can attack, relief by means of resistant varieties will be local or regional. Investigation of this character is being carried on by the United States Department of Agriculture in cooperation with several State agricultural experiment stations, and it is hoped that within a few years valuable information will be available.

"Eradication of Weed Grasses.- Many wild grasses are dangerous carriers and spreaders of stem rust. Among those which rust most commonly and heavily are quack-grass, wild barley or squirrel-tail grass, slender wheat-grass, western wheat-grass, the wild rye-grasses and orchard grass. Some of these grasses are bad and widespread weeds. Where they grow near barberries they almost always rust heavily early in the spring and then serve as centers of infection, from which the rust spreads to other grasses and then to grainfields.....

"Eradication of the common barberry to reduce rust losses.- No one of the methods just discussed will prevent rust entirely, nor can a combination of all of them be depended on to do more than reduce somewhat its amount and destructiveness. The eradication of the common barberry and other rust-carrying species and varieties of barberry gives more promise of success than any other one control measure. The eradication of the barberry cannot be urged too strongly; but the difference between harmful and harmless kinds, the parts of the country in which they are most important, and the results to be expected by removing them should be understood.

"Harmful and Harmless Barberries.- Not all barberry species and varieties are harmful. The common European or high-bush barberry (Berberis vulgaris) is the commonest offender. The purple-leaved barberry is only a variety of the common barberry and rusts just as badly as the green-leaved form. The Japanese barberry, also called the dwarf or low barberry (Berberis thunbergii), on the other hand, does not rust and should not be disturbed, because it not only is harmless but is also a very beautiful shrub.....

"Distribution of the Barberry.- The common barberry is a native of Asia and was brought into Europe 400 or 500 years ago. It was cultivated as a fruit bush for hundreds of years until it was found to spread the rusts of grains. The berries were used for preserves and jellies, and their juice was used for making wine and vinegar. The early colonists brought the bush to North America, and it has been more widely distributed year by year, although recognized as a menace to our grainfields. The bush was popular and the colonists carried the seeds or the bushes themselves with them when they went into new regions. Barberry bushes were planted around the first cabins which the settlers in the Middle West built. There are thousands of bushes in that section which are 40 or 50 years old. Many bushes as old as 60 or 70 years are still thriving. The settlers unknowingly brought with them the greatest enemy to their grain crops. The barberry was there as early as the grain. Nurserymen have been propagating and distributing barberry bushes for many years, although they have discontinued this practice to a considerable extent since they have learned its relation to the rust of wheat and other grains.

"The barberry is especially common in cities, villages, and even in the country districts in the New England States and westward through the upper Mississippi Valley. It is very common in parks, cemeteries, and on public and private grounds, where it has been used in hedges and in clump plantings. Scarcely a village or city of any size in the upper third of the country is without some barberry bushes. The bush is not nearly so popular in the South. Naturally the barberry problem is much more serious in those regions where the bushes are most abundant.

"The common barberry running wild.- The seeds of the barberry are carried
r birds, and the bush has escaped from cultivation to some extent in this way.
n the New England States large numbers of common barberry bushes are found
rowing wild in pastures and fields. Fortunately those States are not primarily
rain-growing States or their problem would be discouraging indeed. But the
arberry has escaped also to some extent in the grain-growing districts of the
iddle West. In Michigan, Wisconsin, Iowa, Minnesota, and other grain-growing
tates some wild bushes occur, although they are not so numerous as to make their
radication impossible. But one trembles to think of the effect on the great
rainfields which furnish us our food if the barberry is allowed to go on spread-
ng until it becomes common in the open fields. The wild bushes which do occur
ow are mostly along the banks of rivers or on rocky hills, ...especially where
here is limestone. Of course, the spreading of the bush in these rocky places
akes it all the more dangerous because it is hard to kill when it has established
ts root system in the crevices of the rocks.....

"Barberry Laws.- Attempts to eradicate the common barberry have been made
or 200 years or more. The movement is therefore not a mushroom growth, but, on
he other hand, it is a gradual and healthy development of a sane idea based on
ears of careful observation and scientific demonstration.

"There is some evidence that a barberry eradication law was passed in Rouen,
rance, as early as 1660. Connecticut, Rhode Island, and Massachusetts all
nacted laws against the barberry between 1726 and 1766. The Connecticut and
hode Island laws were renewed after a period of years, so the results must have
een satisfactory.

"Several European countries passed laws against the barberry shortly after
800. Various States in Germany required the eradication of all barberry bushes
ithin a certain distance from grain fields, while still others required all bar-
erry bushes to be removed within a certain specified time. Denmark, in 1869,
assed a law which gave any person the right to demand the destruction of shrubs
hich were known to spread plant diseases. One of the provinces of France took
dvantage of a law passed in that country in 1888 and carried on an energetic
ampaign against common barberry bushes.

"It is difficult to determine the effect of these old laws, because apparent-
ly many of them were not rigidly enforced. However, in certain localities in
England, on the island of Aero, and, in various other places, the eradication of
the barberry was followed by the disappearance of stem rust. Most of these laws
were passed before the exact relationship between the barberry and the rust was
known. The scientific proof of the relationship was not made until 1865. The
early laws, therefore, are the best possible evidence that farmers realized clear-
ly that barberry bushes increased rust, because at the time that these laws were
passed people were not biased by any scientific statements regarding the matter.

"Recently the movement for barberry eradication has gained great headway.
The reason for this act is that there have been several terrible epidemics of stem
rust. These epidemics stimulated investigation of the exact methods by which the
rust lived over winter and started in the spring. The evidence against the bar-
berry became so clear and convincing that strong sentiment developed for the re-
moval of the bushes.

"For several years a law has been on the statute books of Ontario, Canada, requiring the destruction of the barberry. Recently Manitoba and Saskatchewan have also outlawed the common barberry. In the United States several States have enacted barberry-eradication laws. The Legislature of North Dakota passed such a law in 1917, while during 1918 Colorado, Nebraska, South Dakota, Minnesota, Iowa, and Michigan took legal measures to secure the complete eradication of the common barberry. At this time (April 1919) bills are also pending before the Legislatures of Wisconsin and Illinois. The fight against the barberry, therefore, is on in earnest. These laws have not been in force long enough to determine their effect, but it is safe to say that the bushes will be completely removed from the upper Mississippi Valley within a few years and that rust attacks will become less frequent and less severe."- Stakman, E. C. The Black Stem Rust and the Barberry. U. S. Dep't of Agriculture Yearbook, 1918.

Rust in seed wheat.

Rust spores are sometimes found in pustules appearing on the grains of wheat at maturity. The question arises as to whether this rusty seed will produce rust in the crop growing from it. This question has been answered by Hungerford (16). The summary from his paper is quoted below.

"Summary.

(1) Uredinia and telia of Puccinia graminis tritici Erikss. and Henn. have been found embedded in the pericarp on the hilar end of kernels of wheat and sometimes along the ventral groove as far up as the middle of the kernel. Infected kernels have black hilar ends, and groups of telia appear as shining black specks under either the hand lens or the binocular microscope.

(2) "Only a small percentage of infection was found by examination of the hundreds of samples of wheat from the crops of 1915 and 1916. A little over 1 per cent was the largest quantity found in any sample. The durum wheats were found most commonly infected.

(3) "Infection undoubtedly spreads to the kernel from original infection on the rachis, rachilla, or glumes.

(4) "The germinating power of the seed apparently is not impaired by this rust infection.

(5) "When rusted kernels of wheat were sown in the field, no earlier or more severe rust infection occurred on the resulting plants than on those grown in adjacent plots which were sown either with clean seed or with rust-infected seed which had been treated with the modified hot-water treatment.

(6) "More than 2,500 plants were grown from rusted seed in a specially constructed room in the pathological greenhouse at the University of Wisconsin and no rust infection appeared upon any of them at any time. The conditions of growth of these plants were normal, and they produced plump grain.

(7) "No spread of infection from the pericarp to the young plant was found by examination histologically, although infected seed were germinated under various conditions, simulating as nearly as possible natural conditions in the field.

(8) "No infection appeared upon plants grown from seed which had been covered with viable urediniospores of stemrust before sowing.

(9) "The results of the experimental work here reported indicate that stemrust is not transmitted from one wheat crop to the next by means of infected seed grain. Further, in the writer's judgment, the occurrence of stemrust sori in the pericarp of the caryopses of grains and grasses has no especial significance, but the infection spreads to these tissues just as it does from an infection point in any of the vegetative parts of the plant."- Hungerford, Charles W. Rust in Seed Wheat and its Relation to Seedling Infection. Journal of Agricultural Research. 19: 274-275. June 15, 1920.

Review

Describe the symptoms of the various stages of stem rust on barberry and wheat. Give the complete life history of Puccinia graminis. Is it possible for this rust to be perpetuated in any other manner than by the stage on barberry? In what section of the country especially is the barberry thought to be an important factor in spreading the rust? In what manner does the stem rust cut down the yield? Can the rust be controlled? Are all varieties of wheat equally susceptible to stem rust? Discuss the feasibility of securing resistant or immune varieties of wheat. What does the question of "biological races" or "strains" have to do with resistance and immunity?

References.

(1) Duggar, B. M. Fungous Diseases of Plants, pages 384-395 and 408-414.

(2) Stevens & Hall. Diseases of Economic Plants, p. 365.

(3) McAlpine, D. The Rusts of Australia, pages 1 to 64., and also page 120.

(4) Freeman, E. M. Minnesota plant diseases, p. 159-166.

(5) Ericksson, Jakob. Fungoid Diseases of Agricultural Plants, page 68.

(6) Stakman, E. C. A study of cereal rusts. Minn. Exp. Sta. Bul. 138 (Technical) 1914.

(7) Carleton, M. A. Lessons from the grain-rust epidemic of 1904. U. S. D. A. Farmers' Bul. 219. 1905.

(8) Stakman, E. C. The black stem rust and the barberry, U. S. Dept's Agr. Year Book 1918.

(9) Stakman, E. C. Destroy the common barberry. U. S. D. A. Farmers' Bul. 1058. 1919.

(10) Malhus, I. E. & Durell, L. W. The barberry bush and the black stem rust of grains. Iowa Exp. Sta. Cir. 35. 1917.

(11) Robbins, W. W. & Vasey, H. E. Eradicate Common Barberry. Colorado Agr. Col. Extension Bul. 147. 1918.

(12) Levine, M. N. & Stakman, E. C. A third biologic form of Puccinia graminis on wheat. Jour. Agr. Research 13: 651. June 17, 1918.

(13) Stakman, Levine & Leach. New biologic forms of Puccinia graminis. Jour. Agr. Research 16: 103. Jan. 20, 1919.

(14) Dickson, J. G. & Johnson, A. G. Studies on stem rust in Wisconsin 1918. Wis. Dep't. Agr. Bul. 23. July, 1919.

(15) Stakman, E. C. The black stem rust and the barberry. Yearbook of the U. S. Department of Agriculture. 1918.

(16) Hungerford, C. W. Rust in seed wheat and its relation to seedling infection. Jour. Agr. Res. 19: 274-275. June 15, 1920.

LEAF RUST OF WHEAT

Caused by Puccinia triticina Eriks,

This rust is probably the most common and widely distributed rust in the United States although it does not, as a rule, cause such serious damage as the stem rust does. In the past this rust has also been called Brown Rust and Orange Rust.

The aecial stage of this rust has recently been reported by Jackson and Mains (3) as occurring on Thalictrum.

Laboratory study.

1. Compare the two stages of this rust found on wheat with the corresponding stages of the stem rust. Mount and draw uredospores and teleutospores.

2. If material of the aecial stage is available, compare it with the aecial stage of the stem rust.

References.

(1) Duggar, B. M. Fungous Diseases of Plants, p. 416.

(2) Eriksson, Jakob. Fungoid Diseases of Agricultural Plants, p. 82.

(3) Jackson, H. S. and E. B. Mains. Aecial stage of the orange leaf rust of wheat, Puccinia triticina Eriks. Jour. Agr. Res. 22: 151. October 15, 1921.

B. M. Duggar Diseases of Plants, p. 411.

H. S. and E. R. Miles. Annual stage of the wheat leaf rust of

STRIPE RUST OF GRAINS AND GRASSES

Caused by Puccinia glumarum (Schm.) Eriks & Henn.

The stripe rust, also called yellow rust and glume rust, was first identified in this country in 1915 when a party of plant pathologists were touring the west on an inspection tour. The disease was first identified in Arizona but during the same year it was reported from several other western states, including Oregon. It is now known to occur in all western states from the Rocky Mountain tier westward to the coast excepting New Mexico and including the Black Hill region of South Dakota. It has never been found in the United States east of the 103rd meridian.

This rust is recognized as one of the most destructive of all grain rusts in certain parts of Europe and, although there are no reports of any great damage resulting from it in this country, there is a possibility, though not a probability that it may eventually prove to be as destructive in the United States as it is in Europe. Experimental work with artificial infection has proved that many of our wheat varieties are very susceptible to stripe rust so that with proper weather conditions prevailing for the development of the rust fungus we may expect severe epidemics on susceptible varieties.

There is no known aecial stage of the stripe rust so that the only two spore forms we have to deal with are the teliospores and the urediniospores. Can teliospores infect wheat?

1. Symptoms.- Note that on the more mature host plants both the uredinia and the telia are grouped in long stripes running lengthwise of the leaf or stem. This characteristic is responsible for the common name of the rust. On younger plants the stripe characteristic is not so prominent and the pustules are scattered more evenly over the surface. Occasionally there is a tendency for pustules to grow in more or less distinct rows crosswise of the young leaf. Compare size, color and other characteristics of individual pustules with those of other rusts studied. Draw.

2. Life History.- So far as known this rust is carried over from year to year either by mycelium in living host plants or by uredospores. Examine and draw both uredospores and teleutospores.

Review

How is stripe rust distinguished from other wheat rusts? Discuss its economic importance. State its geographical distribution. How many spore forms are known? How is the disease perpetuated? Write a detailed comparison of this rust with other rusts studied.

References.

1) Carleton, M. A. A serious new wheat rust in the United States. Science N. S. 42: 58.

2) Humphrey, H.B. & A.G. Johnson. Observations on the occurrence of Puccinia glumarum in the United States. Phytopathology 6: 96. February, 1916.

3) Humphrey, H. B. Puccinia glumarum. Phytopathology 7: 142. April, 1917.

4) Hungerford, C. W. Studies on the life history of stripe rust, Puccinia glumarum. Jour. Agr. Res. 24: 607. May 19, 1923.

5) Hungerford, C. W. and C. E. Owens. Specialized varieties of Puccinia glumarum and hosts for variety tritici. Jour. Agr. Res. 25: 363. Sept. 1, 1923.

APPLE AND JUNIPER RUST

Caused by Gymnosporangium juniperi-virginianae Schw.

In the eastern half of the United States, wherever the Juniper (Juniperus virginiana), commonly called red cedar in that section of the country, grows, this rust is of widespread occurrence.

Laboratory study.

a. Telial stage.- Examine some of the "cedar apples" or galls from the Juniper. Note the telial horns. Mount a small fragment of the gelatinous material and find the teliospores. Draw. Does this rust have a uredo stage?

b. Aecial stage.- Observe the aecial stage on apple leaves. Compare with the aecial stage of the stem rust of wheat. Draw.

Text discussion.

The following account of this disease is quoted from Heald (5).

"The Life History of the Fungus.

"The Production of Sori.

"The 'cedar apples' remain dormant during the winter period, but with the first warm damp weather of spring they begin to put out the yellowish or orange projections, or sori. The galls vary in size from about that of a radish seed to two inches in diameter and they may produce from one to several dozen sori.

hen the gelatinous projections are fully formed they give the diseased tree quite striking appearance, especially when the galls are very abundant, and some uninormed people admire these structures thinking they are the flowers of the cedar. n cases of light infection the majority of apples produced will be of medium size, hile in severe infections the greater number will be small as shown in the illusration. In severe infections, the weight of the numerous apples is frequently ufficient to cause a conspicuous drooping of the branches.

"Germination of Teliospores.

"Each gelatinous mass is filled with the two-celled teliospores which very oon begin to germinate in situ. A four-celled promycelium may be produced from ne of the cells of the spore or both may germinate, one generally a little later han the other. In the typical germination, each cell of the promycelium produces lateral process which bears a secondary spore or sporidium, so that eight poridia may originate from a single teliospore. When the germination takes place ith the minimum amount of moisture, the promycelium will be short and compact, ut with increase in the water supply a more elongated growth results. Under favorable conditions the promycelium will be produced and the sporidia matured in 12 to 24 hours. In the initial stages of the germination, the cells of the promycelium have granular orange-colored contents, but with the production of the sporidia they become hyaline and empty and the sporidia alone retain the granular pigmented protoplasm. Some deviations from the normal development are shown when some of the promycelial cells fail to produce sporidia or grow out into somewhat elongated branches, and also when sporidia are produced direct from the side of the teliospore without the previous formation of a promycelium.

"Germination of Sporidia and Infections.

"As soon as the gelatinous sori begin to dry, the sporidia are carried away by the wind and many of them fall upon the surface of adjacent foliage. In case conditions are favorable they germinate, each one giving rise to one or two infection threads. Those spores which fall upon the leaves, young fruits or twigs of susceptible varieties of apples produce infections by the entrance of the infection threads through stomata into the tissues of the host. During a given season, one or more crops of sporidia may be produced, but generally the first crop is much the largest. During the three years in which the disease has been under study at Nebraska, the number of crops of sporidia has been either two or three, with corresponding periods of infection.

"Normal Symptoms of Rust on the Apple.

"The rust shows first on the upper surface of the leaves as pale yellow spots about the size of a pin-head. These spots begin to show a week or ten days after the 'cedar apples' have been in the gelatinous condition. The actual infection, however, takes place during the first 12-24 hours following the drying of the sori, and the remainder of the time is occupied by the development of the fungus hyphae within the tissues of the host, and finally with sufficient growth the external symptoms become evident. As the fungus grows, these spots become larger and assume a deeper color, finally becoming orange-colored. In a few weeks after the first

ppearance of the spots, minute pustules appear in the center of each, and these ater show as small black specks. These black specks represent the openings of he pycnia, which are flask-shaped structures sunken in the tissue of the host. o far as is known at the present time they play no necessary part in the life istory of the fungus.

"With the continued growth of the fungus very little change is noticed in he spots on the upper side of the leaf. The leaf tissue beneath the spots, owever, begins to swell up until finally a cushion or blister one-fourth to one-alf inch in diameter is produced. It is on these cushions of tissue that the ecia or cluster-cups are produced. When the aecia are young, each shows as a inute tubular projection with a brown mass of spores at the bottom, but when ature the wall of the tube may be split and recurved so as to give each a stel-ate appearance.....

Summary of Life History of the Fungus.

"The 'cedar apples' or galls produce sori or become gelatinous during the onth of May. The teliospores germinate in situ, and produce sporidia which nfect the apple during the next few days. The first aecia become mature during he month of July and viable spores are produced in large numbers during this and he two following months. It is during these months that the infection of the edar takes place, but no visible signs of the infection can be noted. The my-ycelium apparently remains dormant during the remainder of the season and the inter period and no 'cedar apple' is produced since its production is dependent pon the growth of the cedar, which is slight or entirely inhibited at this eriod. With the resumption of growth in the spring, the fungus stimulates the issues of the cedar at the point of infection and the result is the formation of 'cedar apples', which first become visible in the month of June. These apples row thruout the summer and fall and at the end of the growing season are practi-ally mature in size and form. They remain dormant during the winter period, and ith the return of conditions favorable to growth produce the characteristic elatinous sori. It will be seen.....that it requires twenty-three months rom the time of infection of the cedar to the period when the resultant galls roduce the gelatinous sori.

"Injury to the Apple and the Cedar.

"The 'cedar apple' fungus grows upon two hosts, the cedar and the apple, and is productive of injury to both. The earlier complaints were largely in connection with the stage passed upon the apple leaves and fruits. The injury to the orchard crop is twofold: first, the direct injury to the fruit; second, the injury to the foliage, which affects indirectly the possibility of fruit production; and third, the injury to the twigs.

"In susceptible varieties of apples the young fruits may be very generally infected. The infection may be localized and result in a deforming of the fruit with a slight reduction in size, or the infection may be general over the surface causing a very marked atrophy of the fruit. The amount of injury which the rust causes to the apple foliage may be very great. In the susceptible varieties

which stand adjacent to cedar trees, almost every leaf may be affected, frequently so seriously that the tree shows a pronounced yellow color even from a distance. In such cases a premature defoliation follows and thus sends the tree into its period of winter rest in a weakened condition."- Heald, F. D. The Life History of the Cedar Rust Fungus, Gymnosporangium juniperi-virginianae Schw. Nebraska Agricultural Experiment Station. Twenty-second Annual Report. 1909. pp. 106-111.

Control.

Wherever practical all junipers within a mile or less of the orchard should be destroyed.

If this is not practical, spraying must be resorted to. It is very difficult however, to obtain good control by means of sprays because a delay of one day after the sporidia are ready for dissemination makes spraying the apple useless. To be effective sprays must be put on as soon as the leaves unfold and the foliage must be kept covered until all danger is past. Ordinarily after the first week in June the danger is past. Lime-sulfur or Bordeaux may be used.

References.

(1) Reed, H. S. The cedar rust disease of apples caused by Gymnosporangium juniperi-virginianae Schw. Va. Exp. Sta. Technical Bul. 9. , 1915.

(2) Reed, H. S., Cooley, J. S. and Craybill, C. H. Experiments on the control of the cedar rust of apples. Va. Exp. Sta. Bul. 203. 1914

(3) Giddings, N. J. and Anthony Berg. Apple rust. West Va. Exp. Sta. Tech. Bul. 154. 1915.

(4) Giddings, N. J. Infection and immunity in apple rust. West Va. Exp. Sta. Bul. (Tech.) 170. 1918.

(5) Heald, F. D. Life history of the cedar rust fungus. Neb. Exp. Sta. Rept. 1909: 105.

(6) Weimer, J. L. Three cedar rust fungi; their life histories and the diseases they produce. Cornell Univ. Exp. Sta. Bul. 390. 1917.

RUST OF INCENSE CEDAR AND POMACEOUS FRUITS

Caused by Gymnosporangium blasdaleanum. (Diet. & Holw.) Kern

This is a common rust in the west wherever the incense cedar (Libodedrus decurrens) occurs. It has all the different spore stages of rusts except urediniospores. It is a heteroecious rust, the telial stage occurring on incense cedar and the pycnial and aecial stages, occurring on apple, pear, quince, and wild pomaceous hosts such as service berry (Amelanchier), and hawthorne (Crataegus).

1. Symptoms

a. On cedar.- Observe telial stage on cedar. Describe and draw. Compare with telial stage of grain rusts.

b. On Pome fruits.- Observe aecia and pycnia on leaves and fruits of various pomaceous hosts. Draw. Compare with aecial stage of stem rust of grains.

2. Life-history.- Examine the various spore forms and tell the part they play in the life-history of the fungus. Draw. Examine section of leaf cut through aecia and pycnia. Draw.

3. Notes.- Write a complete description of this rust including its life history.

References.

(1) Jackson, H. S. A Pacific Coast rust attacking pear, quince, etc. Ore. Exp. Sta. Crop Pest Report 1913-14. P. 204.

(2) Hesler & Whetzel. Manual of Fruit Diseases, page 345.

General Review of Rusts

Give the life history of an autoecious rust. Of an heteroecious rust. In the case of hetereocious rusts, what is one possible means of attacking the control problem, based upon the life history of the fungus? Discuss the problem of resistance and immunity in connection with control of rusts.

RHIZOCTONIA OF POTATOES

Caused by Corticium vagum B.& C. var. solani Burt.

Rhizoctonia, or Black Scurf as it is sometimes called, is one of the most common and widespread of potato diseases, occurring to greater or less degree almost everywhere that potatoes are grown. In Oregon it is one of the worst, if not the worst, potato disease occurring in the state.

1. Symptoms and signs.

a. On the tubers.- Note the black "scabby" growths of resting fungus tissue (sclerotia) sticking to the surface of the tubers. Draw. Sometimes more or less cankerous lesions are formed on tubers. Observe specimens of this type of symptom or look at illustrations in Maine Experiment Station Bulletin 230.

b. On stems.- Observe discolored areas on stems or stolons. Are these lesions above or below the surface of the ground? Do such lesions ever occur on the true roots of the plant? Note also the wefts of mycelium growing up on the stem in some cases. Draw.

c. Little potatoes and aerial potatoes.- In some infected hills only very small tubers are produced. Sometimes small tubers form on the stems above ground. Explain why such small potatoes or aerial potatoes are formed on plants suffering from Rhizoctonia. Draw.

2. Life history of causal organism.

a. The sclerotia.- These structures are instrumental in perpetuating the disease. Tubers bearing sclerotia should never be planted.

b. Basidia and spores.- Sometimes the wefts of mycelium appearing on the stems just above the ground produce large numbers of club-shaped cells (basidia) each bearing two to four spores (basidiospores). These spores probably cannot infect other plants directly but may produce a saprophytic mycelium which may later cause infection. Draw.

Discussion

In the following account of the disease as given in circular 24 of the Oregon Experiment Station, note the different effects which the fungus may have upon the host and especially note the two essential things which must be done in order to obtain a fair degree of control.

"Symptoms.- This disease, which frequently goes by the name of black scurf due to the presence of black sclerotia or resting bodies of the fungus on the potato tubers at harvest time is apparently the commonest and most serious potato disease in the State. The disease manifests itself in a variety of ways, the most important of which are the following:

"Often the young sprouts are attacked and are "burnt off" by the fungus even before they reach the surface of the ground. This may lead to the production of other new sprouts which in turn are also burnt off with the result that a rosette or cluster of sprouts is formed with their tips killed, none of which reach the surface of the ground and are able to produce plants. This is often the cause of poor stands.

"Plants attacked less severely or later in the season may develop lesions or dead areas on the under ground stems and upon the stolons, which so interfere with the normal growth and functions of the plant that the leaves may roll up considerably, small potatoes may form in the axils of the leaves or other convenient places in the top of the plant, or the nodes of the stems may become considerably enlarged and knobby because the starch cannot be properly transported downward, and a large number of small potatoes and a few large knotty ones may be developed underground. This result often gives rise to the term "little potato" disease. The yields in such badly attacked hills are practically worthless for commerce as well as for seed.

"Near the base of attacked plants and extending up varying distances from the ground surface, a gray or white mold-like growth on the surface of the stalks may frequently be found from late June to the last of August. This is the fruiting stage of the fungus in which numerous spores are produced and often has the

appearance of salts collected or the stalks from the soil. The tops of such plants generally show distinct evidence of abnormalities due to the attacks of the fungus.

"Fungus Occurs on Other Plants than Potatoes.- The fungus often attacks other plants than the potato, though generally much less severely, therefore long rotations are essential to hold the disease in check when it is once well established in the soil. The experience of many growers has shown that a three-year rotation will not free the soil of this fungus and that rather heavy losses may be expected from this disease under such conditions. A five-year rotation is much superior and should be used wherever practicable. Because the fungus attacks so many plants even including some wild ones in virgin soil, perfect control of the disease cannot be secured. If reasonable care is taken in the application of the control measures presented in this circular, however, the losses from this disease will on the average not be large.

"Seed Treatment with Mercuric Chloride Necessary to Free Tubers of Disease.- In order to kill the Rhizoctonia sclerotia and mycelium on the surface of the tubers and thus avoid the introduction of the disease into the field with the seed potatoes, potato seed treatment is necessary. Because of the effectiveness of formaldehyde for scab control and the general familiarity of the growers with this method of treatment, and because for some time no better method of treatment was known, formaldehyde has been rather extensively used for treatment of seed potatoes against Rhizoctonia. Experiments conducted during the last ten years in many different states and under different conditions have clearly demonstrated, however, the ineffectiveness of formaldehyde for Rhizoctonia control and the superiority of mercuric chloride over all other methods yet devised for this purpose. The general effectiveness of mercuric chloride treatment for the control of Rhizoctonia has been so clearly and so well shown that no one should hesitate now to use this treatment in preference to all others.

"Comparative Seed Treatment Tests Conducted at Corvallis.- It remains true, however, that quite a number of growers in Oregon are still using formaldehyde for potato seed treatment. This is so probably because they have not yet learned of or fully appreciated the superiority of the mercuric chloride treatment. On account of this fact a seed treatment test was conducted by the Oregon Agricultural College Experiment Station at Corvallis in 1920 to secure records under Oregon conditions that could be presented to Oregon growers.

"Two lots of Burbank seed potatoes selected out of the same bin were used. One lot of tubers was selected as being free of any Rhizoctonia so far as their appearance to the naked eye was concerned. No doubt they did have some Rhizoctonia present but to all ordinary appearances they were free from this fungus. All of the tubers selected for the other lot had Rhizoctonia sclerotia present on the surface.

"These two lots of potatoes were divided, treated by several different methods, and planted on well rotated land to avoid contamination from the soil so far as possible. The formaldehyde solution for treatment was used at the strength of 1 pint of the commercial 40-per cent formaldehyde in 30 gallons of water, which is the standard strength for scab control, and the mercuric chloride was used at the standard strength of 4 ounces in 30 gallons of water. Wooden barrels were

used in making the treatments, since the mercuric chloride reacts with metal and consequently should never be used in metal containers.

"At harvest all the different lots of potatoes were dug separately and the percentage of the tubers bearing sclerotia of Rhizoctonia were determined by counting all the tubers which were apparently free of Rhizoctonia and those which had sclerotia present.....

"Formaldehyde Not Effective for Rhizoctonia Control.- Where Rhizoctonia was present on the seed potatoes, treatment with formaldehyde for 1½ and ½ hours gave crops that were not very much freer of the disease than where the infested seed was planted untreated, the percentages of tubers bearing Rhizoctonia in the crops being 53, 62, and 85, respectively. Formaldehyde then is here shown to be far from effective in controlling Rhizoctonia.

"Mercuric Chloride One and One-half-hour Treatment Gave Best Results.- Treatment with mercuric chloride for only one-half hour gave much better results than were secured with formaldehyde 1½-hour treatment, the comparative percentages of disease being 32 and 53, respectively. By far the best results where diseased seed was used came from the 1½-hour treatment with mercuric chloride, the percentage of disease in the crop in this case being only 17. These figures would indicate that the longer treatment with mercuric chloride should be used wherever possible. There are circumstances under which one might be justified in using the shorter ½-hour treatment, as for instance when the seed is only lightly infested or when the seed is badly sprouted and the injury to the sprouts from long treatment might be excessive. Seed potatoes should be treated while still dormant for then no injury to the tubers will result even when soaked for from 1½ to 2 hours, which is the standard length of time for treatment with mercuric chloride.

"Apparently Clean Seed not Entirely Free of Rhizoctonia.- Where apparently clean seed was planted untreated a considerable amount, 34 percent, of the tubers in the crop was diseased. This is largely accounted for by the fact that the Rhizoctonia fungus was no doubt present, as it is frequently known to be, in the form of indistinct thread-like filaments on the surface of the tubers not easily visible to the naked eye. These filaments, though small and indistinct, are capable of remaining alive on the surface of the tubers and introducing the disease into the new crop provided the seed is planted untreated. This fact illustrates the necessity for the recommendation which the Oregon Agricultural College Experiment Station is urging upon all potato growers in the State; namely, that all seed potatoes regardless of whether they appear to be diseased or not ought to be given the standard mercuric chloride treatment before planting.

"The 'apparently clean' seed when treated with mercuric chloride for 1½ hours gave in the test only 14 percent disease in the crop, which was only slightly less than that secured in the crop from the diseased seed treated in the same way (17 per cent). To some these amounts of disease in these two crops may appear to be excessively high. It should be remembered, however, that Rhizoctonia is a more or less constant inhabitant in our soils and affects a wide range of plants besides potatoes, making it next to impossible to grow a crop of potatoes free from this fungus. No doubt the organism responsible for the disease on these crops came largely if not entirely from the soil. This only emphasizes the necessity for carefully conducting the cropping systems so as to avoid undue accumulation

of this as well as other diseases in the soil, as has already been outlined in previous paragraphs."- McKay, M. B. Potato diseases in Oregon and their control. Ore. Exp. Sta. Circular 24. February, 1922.

Review

Describe all the different types of symptoms of Rhizoctonia occurring on tubers, stems and roots of potatoes. Describe the life cycle of the fungus which causes this disease, especially perpetuation and dissemination. Recommend control measures with reasons. Name other plants which are attacked by the Rhizoctonia disease. (See Ill. Exp. Sta. Bul. 189. 1916).

References

(1) Duggar, B. M. Fungous Diseases of Plants, p. 444, also p. 477.

(2) Morse, W. J. & Shapovalov, M. The Rhizoctonia disease of potatoes. Maine Exp. Sta. Bul. 230. August, 1914.

(3) Shelby, A. D. A rosette disease of potatoes. Ohio Exp. Sta. Bul. 139. 1903.

(4) Peltier, Geo. L. Parasitic Rhizoctonias in America. Illinois Exp. Sta. Bul. 189, 1916.

(5) Duggar, B. M. & Stewart, F. C. The sterile fungus Rhizoctonia. N. Y. (Cornell) Exp. Sta. Bul. 186. January, 1901.

(6) Ramsey, G. B. A form of potato disease produced by Rhizoctonia. Jour. Agr. Research 9: 421. June 18, 1917.

(7) Piper, C. V. & Coe, H. S. Rhizoctonia in lawns and pastures. Phytopathology 9: 89. February, 1919.

(8) McKay, M. B. Potato diseases in Oregon and their control. Oregon Experiment Station Circular 24. February, 1922.

MUSHROOM ROOT-ROT

Caused by Armillaria mellea (Fries) Quel.

Probably few fungi have a wider range of hosts than does the Armillaria mushroom which causes the well-known root-rot disease of so many of our fruit trees and smaller cultivated plants as well as of some forest trees, both coniferous and non-coniferous.

This fungus lives indefinitely on the dead stumps and roots of forest trees and has the faculty of becoming an active parasite on trees and other plants grown on ground, formerly covered with forest, before the fungus has entirely died out.

1. Symptoms or signs.- Examine stumps and roots of trees which have been killed by the disease. Note the killed areas, the mycelial felts under the bark and the "shoe-strings" (Rhizomorphs) clinging to the surface of the roots and resembling small, black rootlets. Draw.

In the field, affected trees may be detected by their sickly appearance and the gradual yellowing of the leaves. Sooner or later mushrooms spring up from the base of the tree or from the roots.

2. Morphology and life history of causal organism.

a. Vegetative phase.- Mount a bit of the mycelial felt from beneath the bark. Of what is it composed? Draw a few threads. Next mount a fragment of a rhizomorph. Of what is it composed? What do you judge to be the function of rhizomorphs?

b. Reproductive phase.- Examine specimens or photographs of mushroom fruiting bodies of the fungus. Draw. Recall to mind the names of the parts of a mushroom; cap (pileus), stem (stipe), gills (lamellae), and ring (annulus). Where and in what manner are the spores borne?

3. Notes.- Write a detailed description of all features observed.

Discussion.

The following discussion is quoted from Barss (2) in the Ore. Exp. Sta. Crop Pest Report for 1911-12.

"Hosts.- From numerous parts of Europe and America it is reported that many forest trees, including various evergreens, the birch, beach, walnut and oak, as well as such fruit trees as the apple, plum, cherry, peach, citrus and olive, besides certain bush fruits and the grape, are attacked by very similar, if not identical, mushroom root rots. Along our Pacific Coast, mushroom root rot is prevalent on many kinds of trees and shrubs. In Western Washington, Lawrence reports its presence among several native trees, both evergreen and deciduous, and mentions that in orchard and field the apple, plum, cherry, gooseberry, currant, blackberry, raspberry and loganberry were found to be badly injured or killed. In California, Horne has reported that it occurs throughout the state, attacking a great variety of hosts. He says that it appears to be capable of attacking almost any plant, in fact, that is somewhat woody and long-lived. In Oregon no careful investigation has been made to determine to what extent native forest trees may be parasitically attacked, but mushrooms like those usually associated with the orchard disease, are frequently found arising from decayed alder, maple, oak, pine and fir trunks, and further search would probably reveal other kinds affected. In regard to cultivated trees, the attention of the department has been more often called to serious damage resulting from attacks on prunes and apples than upon other kinds of fruits, although in some sections the cherry and peach are badly affected. The walnut also has been found parasitized by the fungus. Further investigation is likely to establish its occurrence also on other trees and probably on small fruits in Oregon, since no thorough survey of the disease has yet been made in the state.

"Symptoms.- The symptoms of the disease vary somewhat, depending upon the point at which the tree is attacked, the rapidity or slowness with which the parasitic fungus spreads, and other conditions. It is generally not until the trouble has made considerable progress that the first external evidence of the attack appears in its effect on the health of the tree. The earliest indication is usually a retarded growth, attended by early dropping of the leaves. The foliage generally presents an unnatural appearance. Often this is confined to that side of the tree corresponding with the side of the trunk first attacked. The fruit is usually poor and stunted. A tree may sometimes show such symptoms for two or three years before it finally succumbs, but death frequently occurs during the season in which the symptoms first appear, or during the following year. In rapid attacks, the leaves often become yellow early in the summer. Not infrequently the entire foliage suddenly withers and the whole tree dies in mid-season. Again, death may occur during the winter, in which case the tree fails to leaf out in the spring. Sometimes, but not always, clusters of mushrooms of a light brownish color come up in the autumn around the base of the trunk, or push through the soil above diseased roots. In some cases, this occurs before the tree is totally dead.

As soon as a tree shows the first signs of trouble, it should be examined by digging the soil away from the base, and a search should be made for decayed areas on the trunk and roots, and for peculiar black, rootlet-like branching strands, mostly about the thickness of the lead in a pencil, clinging to the bark. These strands, known as rhizomorphs, are characteristic of the disease, since they are a special form of the fungus which causes the rot. They can be distinguished from the roots of any small plants which may also be present in the soil, by their peculiar irregular method of branching and by the white interior substance which can be rather easily separated from the thin black covering. The dead bark and wood is penetrated by a whitish fungous growth and has a strong mushroom odor. Sometimes rhizomorphs are found on the roots of an entirely healthy tree apparently causing no injury. It is possible that in some instances such strands do not belong to a disease-producing form. This point needs further investigation.

"The disastrous effect of the disease results from the girdling of the main roots and the trunk, and from a destruction of the activity of the sap-wood. This brings about root starvation, a checking of the ascent of water through the wood and, as a consequence, the eventual death of the whole tree.....

"Life History and Method of Spread.- We have very little exact information regarding the way in which this fungus becomes established or how it enters the infected tree. It is thought by most authorities that after the spore germinates, the delicate fungus plant develops as a saprophyte, feeding upon decaying vegetable matter in the soil. After growing for a time in this manner it may form black rhizomorphic strands, which push their way through the soil, and upon coming in contact with the root of the susceptible plant, may possibly bore their way into the healthy bark. It is believed by many, however, that the fungus gains it entrance more often, if not exclusively, at points where the root or the crown of the tree has been injured by cultivation, by the attacks of borers, or by the presence of a crown gall, etc. When once it has entered the tree, it sends out delicate filaments both into the bark, where, given the right conditions, it spreads rapidly, and also, by way of the medullary rays (silver grain), into the wood, where it spreads more slowly. It causes death and decay in the parts

attacked, and with a whitish, felty growth, fills up the spaces caused by shrinkage of the tissues. The bark frequently becomes separated from the wood, and in the space thus formed the fungus spreads out in a fan-like manner. In most cases where the disease has made any considerable progress, this conspicuous feature is noticed upon tearing off the dead bark..... Sometimes the white fungous layer formed under the bark or in cracks of the wood becomes quite thick. While the rot is thus spreading along a root or the base of the trunk, the fungus frequently breaks out from the bark, and, in the form of branching rhizomorphs, creeps along the surface of the root or trunk, working its way out into the soil. It is probable that these strands may enter the tree again at new places, thus spreading the attack, or coming in contact with the outlying roots of other trees close at hand, may enter them, and thus cause further extension of a diseased area in the orchard.

"The disease spreads fast from the point of attack up and down the root or trunk, apparently working most rapidly in the cambium region. Where a root is attacked, little effect on the tree is at first noticeable, perhaps for several seasons, until in the course of time the decay reaches the trunk, whereupon the activity of the fungus may quickly cause the girdling and death of the tree. Where the first point of attack is at the crown, or near it, the progress of the disease is more rapid than where a root is the initial point of infection. The decay rarely extends more than a foot or two above the ground and is not usually detected on the trunk at first unless the bark is stripped off. After a tree is dead, the growth of the fungus is checked by the drying out of the parts above ground, but it may continue to live in the underground parts, if there is sufficient moisture present, until the roots and stump are entirely decayed. From these rotting roots rhizomorphs run out into the soil and form a means of spreading the disease to the roots of unaffected trees.

"When the decay has progressed sufficiently and the fungus is thereby richly supplied with reserve nutriment, the fruiting bodies or mushrooms may be formed. In their young condition these are edible. Abundant moisture is necessary for their development, consequently they are not met with, as a rule, till the fall rains have begun. The clusters of mushrooms usually appear at the point where the trunk enters the earth and may encircle its base. Often they rise through the soil from a diseased root or they may be attached to rhizomorphic strands connected with crown or roots.....

"Control Measures.- ..... The prevalent,..... idea that buried roots of forest trees in newly cleared land are a dangerous source of infection for young orchard trees, has led to the suggestion that all roots and stumps be carefully removed from the earth in clearing, and that before planting trees, the soil be devoted to other than orchard crops for several years until the root-rotting fungus has had a chance to die out.

"Evidence that much of the infection takes place where trees are injured by cultivation leads to the suggestion that cultivation close to the tree be done with greatest care to avoid injury. At the same time thorough cultivation has been advised in order to establish good aeration of the soil, since this condition is supposed by some to be unfavorable to the progress of the disease while it undoubtedly promotes the vigor of the tree.

"When trees are once attacked by the fungus, death, almost without exception, s only a question of time. It seems useless, therefore, to retain in the orchard rees which will be worthless to the owner and which may become a source of infec- ion for other trees. It has consequently been considered best to grub up at once ny tree discovered to be diseased and to remove it from the orchard, taking out t the same time as much of the root system as possible. It has also been advised ot to plant another tree in its place for at least three years.

"If it should seem desirable to retain temporarily a tree known to be diseased, t would perhaps be safest to dig a trench about two feet deep around the tree near he tips of the roots in order to prevent the rhizomorphs, which rarely occur eeper than this, from spreading to other trees. Throw the dirt inside the ditch. here conditions are such that the disease appears to be spreading rapidly from ree to tree, the heroic method of uprooting healthy trees in advance of the zone f infection has been suggested; but as far as we know, the disease rarely assumes uch a condition in Oregon orchards as to warrant so drastic a method of treatment.

"On account of the possibility of infection from spores produced by the mush- ooms, all fruiting bodies appearing at the base of diseased or dead trees, or in ther places in the orchards and fields, should be collected while still in the utton stage and destroyed by burning. Knocking them over does not prevent spore ormation.....

"Those who possess sufficient patience and skill, and are willing to devote he necessary time, may be able by the following method to save individual trees hat are not too far gone when the disease is discovered. This method has been sed in one Oregon apple orchard with considerable success. Whether it can be pplied as well to other kinds of fruit trees remains to be seen. At the first sign of unhealthy foliage the tree is examined for root rot. Where this is found, he earth is removed from the base of the tree and the main roots are exposed for a foot or more. The soil is also removed from beneath the roots. The air and sunshine have access in this way to the crown and root bases. The dead portions of the bark are carefully and completely removed from trunk and roots. If a root is girdled, it is taken out entirely. After all the diseased parts of the tree are cut out, the exposed surfaces are washed with some disinfectant such as Bordeaux mixture."- Barss, H. P. Mushroom Root Rot of Tree and Small Fruits. Oregon Agricultural College. Biennial Crop Pest and Horticultural Report. 1911-12: 226-233.

Review

Describe all symptoms of mushroom root-rot. Describe the structure of the mycelial felts formed under the bark of the host, and also the structure of the rhizomorphic strands. Describe the complete life history of the fungus. What part do the rhizomorphs play in perpetuation and dissemination? The mushroom fruiting bodies? Discuss control measures.

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References

1) Duggar, B. M. Fungous Diseases of Plants, p. 473; also p. 471.

2) Barss, H. P. Mushroom root rot of trees and small fruits. Ore. Exp. Sta. Crop Pest Report 1911-12. Page 226.

3) Wilcox, E. M. A rhizomorphic root-rot of fruit trees. Oklahoma Exp. Sta. Bul. 49. 1901.

WOOD ROTS

Caused by various species of Basidiomycetes

Perhaps few people realize fully the enormous losses due to wood decay. Wood does not "just rot" without any cause. All decay in wood is caused by some kind of an organism.

By far the greater part of all wood-rotting organisms belong to four families of the Basidiomycetes, namely; (1) Polyporaceae (Pore fungi); (2) Hydnaceae (Tooth fungi); (3) Agaricaceae (Gill fungi); and (4) Thelephoraceae (Smooth fungi).

As a matter of convenience, in this exercise we shall take up the study of wood rots under two heads: (1) rots of forest trees, and (2) rots of fruit trees. As a matter of fact, there is no difference between these two groups, because all of the fungi which attack fruit trees can be found on forest trees in the vicinity and undoubtedly are transferred from the forest trees to the orchards.

A. Wood rots of forest trees.

The majority of the species of fungi which attack forest trees belong to the family, Polyporaceae, but several species belonging to the other three families mentioned above are of considerable importance. Fungi may cause heart-rot of living trees, heart or sap-wood rot of dead or fallen trees, or decay of manufactured lumber.

Some of the most common wood rots are listed below.

I. Caused by Polyporaceae.

(1) Pecky wood-rot or ring-shake caused by Trametes pini (Ring scale fungus).
(2) Red-brown sap-wood rot caused by Fomes pinicola (Red belt fungus).
(3) Brown checked wood-rot caused by Polyporus sulphureus (Sulfur fungus)
(4) Red-brown root- and butt-rot caused by Polyporus schweinitzii (Velvet-top fungus).
(5) White heart-rot caused by Fomes igniarius (False tinder fungus).
(6) Brown heartwood-rot caused by Fomes officinalis (Chalky quinine fungus).

(7) Brown heartwood-rot caused by Trametes carnea (Pink-bracket fungus).
(8) Brown wood-rot of trees and lumber caused by Lenzites saepiaria.

II. Caused by Hydnaceae.

(1) Stringy brown rot caused by Echinodontium tinctorum (Indian paint fungus).
(2) White, wet heartwood-rot caused by Hydnum erinaceus.

III. Caused by Agaricaceae.

(1) See under "mushroom root-rot".
(2) White streaked sapwood-rot caused by Pleurotus ostreatus (Oyster fungus).

IV. Caused by Thelephoraceae.

(1) Probably the most common and conspicuous wood-rotting fungi of this family belong to the genus, Stereum, of which there are many species.

B. Wood-rots of orchard trees.

Some of the fungi which are commonly found on forest trees also frequently occur on orchard trees. One seldom finds a mature orchard of any kind of fruit in which there are not at least a few trees in various stages of decay or perhaps with one or more of the main branches broken down at the crotch because of the ravages of wood-rotting fungi, and in many instances a very large percentage of trees is more or less damaged by rots.

A majority of the species of fungi causing rots in fruit trees belong to the family of "Pore Fungi" (Polyporaceae) as was found to be the case with forest trees. Occasionally, however, "gill" forms (Agaricaceae) and "tooth" forms (Hydnaceae) also are found on fruit trees as well as on forest trees.

It has been noted that the fungi causing rots in forest trees enter through wounds of various kinds, usually broken off branches or fire wounds. Rots of fruit trees also enter through wounds, but in this case the wounds are usually man made and the rots which start in this manner are therefore more susceptible of control.

Following is a list of some of the fungi which cause rots in fruit trees:

(1) Heart rot of fruit trees, especially peaches and prunes, caused by the Pink Bracket fungus, Trametes carnea.
(2) Heartwood and sapwood rot caused by Polystictus versicolor. Attacks all kinds of trees.
(3) A rot similar to (2) caused by Polystictus hirsutus.
(4) A rot of Cherry trees caused by Irpex lacteus.
(5) A rot caused by the Red Belt fungus, Fomes pinicola.
(6) A white heart-rot caused by Fomes igniarius.
(7) A decay caused by Schizophyllum commune, a fungus which is probably mostly saprophytic or weakly parasitic.

Laboratory study of wood rots.

1. Examine wood rotted by any or all of the various fungi mentioned above and note the characteristic effect in each case. Sketch and describe.

2. Observe sporophores or fruiting bodies of each species of fungus. Note size, shape, color and location on host. Note character of spore bearing surface. (In the pore fungi, the spores are borne on basidia which line the inner surface of the pores. In the tooth fungi, each tooth is surrounded by spore-bearing basidia. In the smooth fungi, the spores occur on the smooth, under surface of the sporophore. In the gill forms spores are borne on both sides of each gill.) In sections of sporophores note internal structure of the fruiting body. Draw.

3. Examine sections of decayed wood under the microscope. Note mycelium in the tissues. What effect on the wood is evident? Draw.

4. Write a detailed description of each of these wood rots, including rotted wood and sporophore of the fungus. Discuss the life cycle of the fungus, especially dissemination and infection. How can infection be avoided? Discuss the proper method of pruning and of treating wounds to prevent rots in fruit trees. Give the formula for making Bordeaux paste.

Discussion.

A. Rots of forest trees.

In the U. S. D. A. Yearbook for 1900, Von Schrenk discusses the problem of wood-rots in forest trees as follows:

"The fungi which are of particular interest to the lumberman and forester are those which render the wood valueless. Of these, there are a large number, some living in the wood of the roots, others in the wood of the trunk and branches. Some are confined to the dead heartwood, while others can grow in both heartwood and sapwood. The fungus threads penetrate the wood cells in all directions, and gradually destroy them. This weakens the roots of the trunk, and frequently results in the overthrow of the tree during some windstorm, or when the tops are laden with snow..... The trunks of some trees are attacked in the tops, and in that case the trees may remain standing for many years with their heartwood completely decayed. The presence of such fungi is not generally detected until the trees are cut down. Lumbermen have, however, learned to recognize some of these trees to be diseased by various signs, such as the presence of resin accumulations, or of the fruiting bodies, called conchs, or punks.

How trees are Infected

"Plants in general have numerous natural means for protecting themselves against the attacks of fungi, such as the cutinized, or skin-like, surfaces of the leaf, the thick bark of the tree trunks, and the exudation of gums and resins, which close wounds against influences from without. During the early life of a

ree these means are more effective than later on, when the vigor of the tree is educed. Wherever a break is made in these natural barriers, insects and fungi re sure to enter. The two parts of the trunk, the living and dead parts, are ttacked by different kinds of fungi, and when wounded they react in very different ays. Wounds made in the live parts are quickly covered with gum or resin, ollowed by their complete healing over by a layer which develops from the edges. n the tender parts of leaves and branches a cork layer frequently forms below the ounded areas, which makes the entrance of fungus spores and threads more difficult, ecause of this rapid healing, one finds but few destructive fungi entering the ive parts. The dead part, on the other hand -- that is, the inner portion of the ree comprising the heartwood -- has of itself no means of excluding the spores or ilaments of fungi. Its sole protection lies in the living layer which surrounds t on all sides. When the latter is broken so as to expose the dead inner part or only a small space, the exudation of gum or resin from the living parts may uffice to protect the dead parts particularly when the tree is young. In those rees which do not make gum or resin, a peculiar brown gum-like substance forms in he wood cells when exposed to the air, which is supposed to protect the exposed arts against fungus attack. Moreover, one finds that the heartwood of some trees s protected against these fungi, probably because of some peculiar chemical com- ounds which are contained in the wood cells. The wood of the Cypress (Taxodium istichum), Red Cedar (Juniperus virginiana), and Redwood (Sequoia sempervirens) s an example of this kind.

How wounds are formed

"In the primitive forest wounds are made in the trunks of trees by deer and ther browsing animals, which eat the bark and newer wood. Bears are fond of ulling long strips of bark from the various cedars in order to get at the sugary apwood. Wounds of this kind heal rapidly, and when they give entrance to spores, hese are usually of the parasitic kind that do but little harm. Birds, particular- y woodpeckers, make large holes in living trees, which oftentimes give entrance o wood-destroying fungi. Boring insects make holes in the bark and wood of stand- ng trees which result not only in the death of the tree, but hasten the entrance f fungus spores. The roots are injured by burrowing animals, particularly by oles. The wounds made by animals are insignificant, however, when compared with he wounds caused by the breaking of large branches. As a tree grows older the larger branches are composed of heartwood and sapwood, and when such a branch is roken off by the wind or the snow the heartwood is exposed. Many trees speedily over the broken branch stub with a callous layer, but when the break occurs far ut from the trunk this may never be accomplished.....

"In the meantime the spores of fungi germinate on this stub; the hyphae of the fungus grow into the dead wood of the branch and through it into the heartwood of the main trunk. While the tree is young the natural pruning which takes place in a dense stand causes many branches to break off, but these are generally small, and, furthermore, they break off close to the trunk because of the constant pres- sure of the callus at their bases.

"Boring insects assist in making passageways for the fungus threads through layers which they could not cross. The old resin-infiltered branch stubs of coniferous trees are not penetrated by fungus threads. Many borers enter these dead branches, however, and pass down them into the trunk.....

Age at which trees are infected

"Young trees have few wounds, and these heal rapidly. As the tree grows older the chances for infection become greater every year, and after a certain age has been reached one may say that almost all trees are diseased at some point, a fact well known to lumbermen. It will be of importance to determine what this age is for the different kinds of timber, for it is often possible to take this into account in the harvesting of a timber crop. When trees have reached this age they may be considered ripe, and the yearly growth and consequent increase in value will be overbalanced by the almost certain chances of destruction by one or another of the wood-destroying fungi.

How the Fungi Destroy the Heartwood

"The fungi are a low class of plants which require certain substances to sustain their growth, just as the higher green plants do. While the latter take comparatively little from the ground in the way of food, the fungi are entirely dependent upon their host plants. By means of certain complex chemical substances they are able to feed upon the plant on which they are growing, utilizing the contents of living cells, or their walls, or both. The wood-destroying fungi extract certain elements from the walls of the wood fibers, which give rise to complex chemical changes in these fibers, differing according to the fungus. Some of them extract the substances which bind the individual fibers to one another, leaving them as separate shining white cellulose fibers, while others extract the cellulose and leave a brittle brown mass resembling charcoal. These changes may go on throughout the wood, or may be confined to local areas. The changes brought about by the fungi are popularly spoken of as rotting, and whatever their nature, they destroy the mechanical properties of the wood. When the decay is induced by fungi entering at the roots, it gradually spreads up the trunk, and in course of time the latter becomes weakened to such an extent that it breaks, usually at a point some 10 feet from the ground. The tree may be perfectly healthy and alive on the outside when it falls, but it dies rapidly after the fall, so in one sense the fungi destroying the heartwood may be said to kill the tree.

"From the standpoint of the lumberman these fungi ruin the wood for commercial purposes, and the amount annually so destroyed can only be guessed at..... ...

Fruiting Organs

"After the vegetative filaments of the fungus have extracted a sufficient amount of nutritive material from the wood they grow outward to form the fruiting organ which is to produce the spores. These organs vary in form and in their mode of occurrence. Many of them are the well-known punks, or conchs, toadstools, or frogstools, found on the trunks.....

"The spores are formed on fine filaments in long tubes or on the spines and gills found on the lower surfaces of the fruiting body. When ripe they are discharged in clouds; they float off through the air and lodge in the crevices of the bark and in wounds. When one considers the countless thousands discharged by one sporophore every year it is not to be wondered at that the chances for infection are very great.

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[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

"The fruiting bodies may be one or more years old. In those which grow to e more than one year old a new layer is added on below the old ones, and in this ay a particular individual may grow to be fifteen or twenty years old. The number f fruiting bodies on a trunk varies. The fungus causing the white rot of the oak sually forms but one fruiting body; so also the one causing pin rot of the Incense edar (Libocedrus). The majority form many on one trunk, increasing in number from ear to year.....

"There are a number of fungi which grow on dead timber which are of importance o the engineer and constructor. The spores of these fungi are deposited on sawed imber in the forests, and when placed under favorable conditions they germinate nd bring about a process of decay which is very much like that found in live imber. Absence of sufficient air circulation, presence of moisture, and equable emperature are conditions which favor their growth. This is very marked in the ase of railway ties, of which many thousands are destroyed by these fungi every ear. The position of the tie in the roadbed, surrounded by earth or cinders, rings about conditions on the lower side of the tie extremely favorable to the rowth of these fungi, which gradually rot the wood, and ultimately form the fruit- ng bodies (the conchs, or punks) on the ends of the ties. In buildings, other ungi attack large beams wherever any moisture accumulates, which allows their pores to germinate."- Von Schrenk, Herman. Fungous Diseases of Forest Trees. earbook of Department of Agriculture 1900: 200-210.

B. Wood Decay in Orchard Trees

The following is quoted from a California circular (9) and applies especially o California conditions, but is undoubtedly true to a large extent for Oregon also.

"Source of Infection.- The fungi which cause wood rot in the orchard can be ound abundantly in the native growth, especially along wooded streams. Old orchards also become sources of infection. The destruction of sporophores in a ell-isolated orchard would reduce the number of spores present, yet birds or insects might easily carry spores for several miles.

"Susceptibility of Different Fruit Trees.- Observations suggest the following rder of susceptibility of orchard trees to decay: Peach, cherry, apple, plum (including prunes), apricot, walnut, pear, olive, and citrus trees. Olive and citrus trees will probably become hollow without showing the more violent effects or rot, such as sappy bark of apple, but even they must be to a certain extent crippled.

"Prevalence of Wood Decay in Orchards.- It is well recognized that some decay- ing trees occur in all sections and in nearly all orchards, especially of stone fruits and apples. The actual percentage of decayed trees can evidently be deter- mined only by careful examination of large numbers of representative orchards, in which either every tree should be examined, or a certain number of trees taken in a way to represent the whole. A number of examinations of both kinds have been made by Mr. W. W. Thomas for stone fruits. In the districts examined in central California it appears that nearly fifty per cent of all bearing stone fruit trees are positively infected with wood decay. Of the remainder not more than about two per cent are free from large cracked wounds which may already be infected or are

in great danger of infection. Orchards which are not very old can be found in which every tree has decay. Grafted-over trees usually show a very high proportion of decay, frequently approaching a hundred per cent.....

"Professor Wickson states that the peach orchards in central and northern California are not old enough to mark the normal life of a peach tree, and the best authorities seem to agree that fifty or sixty years will not find the peach tree aged except by the entrance of decay into the wood. Yet the usual length of life of a peach orchard in California is not half this period."- Horne, W. T. Wood Decay in Orchard Trees. University of California Agricultural Experiment Station Circular 137: 3-5. 1915.

Wood-decay in Oregon Orchards

S. M. Zeller (14) of the Oregon Experiment Station has made a survey of some of the orchard districts in this state and finds the wood-rot situation serious. Some of his conclusions are given below.

"The prevalence of wood decay in orchards is sure to be associated with the prevalence of large wounds on the trees. Any exposed wood surface of considerable area is sure to become a seat of infection if it remains untreated. Out of a survey of 929 prune and peach trees which showed only ordinary care there were 844 trees having pruning cuts or other wounds over two inches in diameter. Of the 929 trees examined there were 819 showing positive evidence of heart rot and these were all trees having wounds over two inches in diameter. That is, 97% of the trees with large wounds were found to be decayed. In orchards where wounds show careful treatment the percentage of visible wood-decay infection seldom exceeds 3%.

"Results of Wood Decay are Deleterious.- There are many deleterious results of wood decay. It is first a source of weakness to the framework of an affected tree. A tree may have been so pruned that its scaffold branches bear a relation to each other such as to produce the strongest type of tree, yet decay of the wood in such a structure becomes very serious when heavy crops are borne or strong windstorms occur.....

"The encroachment of decay upon the sap wood also cuts down the increment of productive wood as well as the productive longevity of the tree.....The permanent success of future crops from Oregon orchards depends upon the retention of old trees of great vitality and stability. Trees devitalized by internal decay are more susceptible to various fungous diseases, and observation has revealed that they are also less able to withstand severe winters.....

Preventive Measures

"Among the measures which should always be taken to prevent wood decay are the following:

(1) Young trees should be so pruned that large cuts will be reduced to the minimum in future pruning operations.

[illegible] of [illegible]. [illegible] has decay [illegible] decay, [illegible] per cent [illegible]

[illegible] that [illegible] seem to agree that fifty or sixty [illegible] by the entrance of decay into [illegible] orchard in California is not [illegible]

[illegible]-decay in Oregon Orchards

[illegible] (14) of the Oregon Experiment Station [illegible] in their state [illegible] clusions are given below.

[illegible] of wood decay in orchards is [illegible] large wounds on the [illegible] become a seat of [illegible] prune and peach trees which [illegible] pruning cuts or other wounds over two inches [illegible] there were 810 showing positive evidence of [illegible] having wounds over two inches [illegible] wounds were found to be decayed. In [illegible]

Wood Decay [illegible]. There are many [illegible] it is [illegible] have been so pruned [illegible] to produce the [illegible] type of tree, [illegible] become very serious [illegible] heavy crops are borne [illegible]

[illegible] of decay upon the sap [illegible] as well as the [illegible] from Oregon [illegible] vitality and stability. [illegible] to various fungous diseases, [illegible]

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[illegible] should be [illegible]

(2) In older trees all pruning cuts of any size should be made close to the parent branch or trunk so that the bark will not die back from the cut and leave a projecting stub of dead wood..... .
(3) Wounds by orcharding machinery should be avoided.
(4) All dead wood should be trimmed out.
(5) Dead bark and rough portions of cankers and other wounds should be cleaned away.
(6) All pruning and accidental wounds should be treated with an antiseptic after they have been disinfected by the removal of all rough portions and all indications of infection.

Antiseptics for Wounds

"There are many antiseptics for tree wounds which have been recommended and applied with various degrees of satisfaction. The preparations used for covering wounds may be grouped as "air-tight" or "air-porous" coverings. Asphalts, lead paints, and grafting waxes are some-times used as "air-tight" coverings, while some coal-tar products and bordeaux paste are examples of the "air-porous" coverings. There have been objections to both forms of wound dressings. Volck's (Cal. Com. Hort., Monthly Bul. 6: 80-89, 1917) objection to the "air-tight" covering is that the sap pressure back of such a coating will rupture it and there will be enough of the sap retained behind the coat to keep the wood moist and to insure infection by wood-rotting fungi. Bordeaux paste is the one "air-porous" covering which meets with the greatest approval. The one objection to this material is that it is gradually weathered away and its application must be repeated annually.

"For some time the plant pathologist at the Oregon Experiment Station has been recommending the use of Bordeaux paste as an antiseptic for tree wounds. The commercial bordeaux paste, or bordeaux dust made up as a smooth paint by mixing with water, furnish the most practicable and most easily prepared pastes. However, if the grower prefers to mix his own paste the following formula may be of service:

Formula for Bordeaux Paste

"Bluestone Solution.- Dissolve 1½ lbs. of bluestone (copper sulfate or blue vitriol) in one gallon of water in a wooden, earthenware or glass vessel. This is best done by suspending the chemical in a bag of loosely woven cloth, or burlap, at the top of the water, or by pounding the lumps into small bits and dissolving by the use of hot water. This solution attacks metals (except copper) very actively.

"Lime Paste.- Slake three pounds of quicklime with one gallon of water, in the ordinary manner.

"Mix in Equal Parts.- When the lime is cool enough, equal parts of the bluestone solution and the lime paste should be mixed together thoroughly. Mix only enough at one time for a day's use. Apply like whitewash with a brush. The stock bluestone solution and stock supply of slaked lime can be kept indefinitely if not mixed together, provided water is added from time to time, to keep the solution up to the original volume.

"If bordeaux paste is used alone its application should be repeated every all.

"In a recent communication Professor H. S. Fawcett, of the California Citrus xperiment Station informs me that in southern California they have found bordeaux aste to be better than other wound coverings tried there. Where grafting wax, or example, was used as a complete covering for large winter-injury wounds there as much more subsequent heart rot apparent than in similar cases where bordeaux aste was used. Fawcett says: 'The healing of the cambium was better and more apid under the grafting wax than under the bordeaux, but this temporary advantage as many times counteracted later by the more rapid development of the rot in the ood.'

"We believe that Volck has devised a combination which will prove very satis- actory for those that desire in addition to an "air-porous" covering over the xposed wood an "air-tight" covering over the cambium to hasten the initial heal- ng. He has recommended the use of a combination of asphaltum and paraffin for he treatment of the cut bark, lapping it over the cambium and edge of the wood, ith bordeaux paste and copper tacks or nails as antiseptics for the wood. The sphaltum-paraffin wax is made by melting together and thoroughly mixing eight arts of asphaltum to two parts of paraffin by weight. It is applied to the argin of a large wound so that it covers the cut edge of the bark and laps over nto the sap wood enough to protect the bark from drying out until growth begins o cover the wood: This coating is applied warm immediately after the cut is ade. The exposed wood is then coated with bordeaux paste and driven full of opper nails. The bordeaux paste contains enough copper in a soluble form so that t gives temporary prevention of wood-decay infection until the copper nails or acks which, through their gradual solution by the acids of the wood, produce a ore permanent antiseptic condition. It is well in this treatment, Volck suggests, o repeat the application of bordeaux paste to fill up the cracks which later evelop in the wood as it dries until the action of the copper nails has become ffective. On very large wounds Volck recommends the use of copper-wire screen astened down to the wood by copper nails. The bordeaux paste sticks much better o such a screened surface."- Zeller, S. M. Wood Decay in Orchard Trees in regon. Third Crop Pest and Horticultural Report Oregon Agricultural College xperiment Station 1915-20: 132-136. January 10, 1921.

Review

Name some of the worst wood rots of coniferous trees. Of non-coniferous rees. Name some fungi which rot both coniferous and non-coniferous trees. escribe the characteristics of each type of rot studied. Describe the fruiting odies of each wood-rotting fungus studied. Describe the appearance of sections f diseased wood under the microscope. How does the fungus act on the wood? escribe the complete life cycle of a wood-rot fungus. Discuss losses from this ource. Discuss the problem of control.

References

(1) Duggar, B. M. Fungous Diseases of Plants, pages 452-469.

(2) Atkinson, G. F. Studies of some shade and timber destroying fungi. N. Y. (Cornell) Exp. Sta. Bul. 193, 1901.

(3) Schrenk, H. von.- Diseases of deciduous forest trees. U. S. D. A. B. P. I. Bul. 149. 1909.

(4) Meinecke, E. P. Manual of forest tree diseases common in California and Nevada. Issued Feb. 14, 1914 by Forest Service, U. S. D. A.

(5) Hedgcock, G. G. Notes on some diseases of trees in our National Forests III. Phytopathology 3: 111. April, 1913.

(6) Hedgcock, G. G. Notes on some diseases of trees in our National Forests II. Phytopathology 3: 111. April, 1913.

(7) Schrenk, H. von. The decay of timber. Bureau of Plant Industry, U. S. D. A. Bul. 14, 1902.

(8) Rankin, W. H. Manual of Tree Diseases.

(9) Horne, W. T. Wood decay in orchard trees. California Exp. Sta. Cir. 137. 1915.

(10) Hesler and Whetzel. Manual of Fruit Diseases, p. 72.

(11) Zeller, S. M. Heart rot in orchard trees. Oregon Countryman. Vol. 12, No. 3, page 10. Dec., 1919. (This article includes a formula for Bordeaux paste).

(12) Selby, A. D. Tree fillings and wound dressing for orchard and shade trees. Ohio Exp. Sta. Cir. 150. 1915.

(13) McMurran, S. M. Preventing wood rot in Pecan trees. U. S. D. A. Farmers' Bulletin 995. 1918.

(14) Zeller, S. M. Wood decay in orchard trees in Oregon. Oregon Experiment Station Crop Pest and Horticultural Report. 1915-1920, p. 132. 1921.

(15) Volck, W. H. Pruning fruit trees, with special reference to the apple. Monthly Bul. Calif. Comm. of Hort. 6: 80-89. 1917.

CHAPTER XXI.

Diseases caused by Fungi-Imperfecti.

We have already noted that many fungi have an asexual or conidial stage to which we have sometimes referred as an imperfect stage. The downy mildew of onion, the brown rot of stone fruits and the powdery mildews may be cited as examples of diseases caused by fungi which possess a conidial or imperfect stage in addition to perfect stage. This latter stage consists of an oospore in case of the downy mildew of onion, and of ascocarps bearing asci and ascospores in case of the brown rot and the powdery mildews.

It so happens that a great many fungi possess only a conidial or imperfect stage in their life history. It would perhaps be more accurate to say that only an imperfect stage is known. Some fungi which formerly were thought to possess only an imperfect stage have more recently been discovered to possess a perfect stage as well. It was as late as 1902, for example, before the perfect or cup stage of brown rot was found to be just one stage in the life history of the brown rot fungus. Until the brown rot fungus was known to have a perfect stage, the conidial stage was classified as Monilia in the Fungi Imperfecti. There are still thousands of forms of fungi known only in the conidial or imperfect stage All of these have been classified in various orders, families, genera and species under the class, Fungi Imperfecti.

Following are several plant diseases caused by various species of the imperfect fungi.

FUSARIUM WILT OF POTATOES

Caused by Fusarium oxysporum. Schl.

Fusarium wilt is one of the serious potato diseases in Oregon and is not uncommon in many other parts of the country.

1. Symptoms.

a. In stems.- This disease is characterized, at times, by a sudden wilting and dying of the plants while at other times the plants may go down more slowly. A cross section of the lower part of a diseased stem will show a browning of the vascular elements running up the stem. Examine diseased stems and show this feature in a drawing.

b. In tubers.- The fungus which causes a browning of the vessels in the main stem may run out in the vessels of the branch or stolon on which the tuber is growing and finally enter the tuber at the stem end. In case this happens, a section across the tuber near the stem end will show a circle of brown spots. These brown areas are the vascular bundles through which the fungus travels in

ntering the tuber. In some cases, however, the fungus may be present without ausing discoloration. It is in tubers of this sort that the disease may be dis-eminated, but the fungus is usually found living in soils even where no potatoes ave been grown. Examine specimens and sketch.

2. Morphology and life history of causal organism.

Examine cultures of Fusarium oxysporum. Mount and draw some of the pores.

The disease may be carried over in diseased tubers but new infections are ore apt to come from the fungus which is usually present in most soils as a aprophyte.

Discussion

The term "Fusarium-blight" has been applied to this disease by MacMillan but he name "Fusarium-wilt" will doubtless long persist. Below are some quotations rom MacMillan's article on this disease as it occurs in certain potato-growing ections of Colorado.

Fusarium-blight of Potatoes under Irrigation

"The Irish potato (Solanum tuberosum) has been one of the most profitable crops grown in the Greeley district of northern Colorado..... In 1915 a laboratory was established at Greeley for the study of potato troubles, but since that year the yearly losses have been light. Fusarium-blight has been present, however, as a conspicuous malady.

"Fusarium-blight, or potato-wilt, is well recognized in nearly all the potato-growing regions of the country, except in the extreme Northeastern States. It is caused most frequently by the fungus Fusarium oxysporum Schlect., though other species of this genus have been found involved.....

"Description of Diseased Plants.- The common manifestations of the disease are a wilting and rolling of a few leaves, followed quickly, slowly or intermittently by the wilting of the remainder and usually the premature death of the foliage. Occasionally a single leaf will wilt, turn yellow, and die, whereas the remainder of the plant may continue healthy throughout the season. Frequently one of the two or more stems of a hill wilts and dies, while the others remain turgid and healthy. The time of appearance varies greatly. It may be noticeable when the first leaves appear or at any time throughout the season up to maturity. A faint lightening in the color of the plant may indicate a gradual attack, and when the attack is severe, a large plant may pass from health to complete collapse in two days. Late in the season, three or four weeks before frost, plants turgid and unwilted, are found upon which all the upper leaves are rolled. They are a little lighter in color than normal plants, and have often been designated by the uninitiated as being diseased by leafroll..... These plants may continue to lighten in color, the leaves to roll, and gradually to die. These conditions are due to the presence of Fusarium mycelium in the vascular tissues of the stem.....

on examining plants in the earlier stages of wilt, the stem and roots are ually found clean and apparently healthy, but the seed piece has rotted, and mains as a wet, jelly-like mass. Later in the season the seed piece practically sappears, and the roots and stem may become blackened and decayed. At first the ems may be wet and slippery, but in time they become dry, brittle, and friable. e main and lateral roots of plants having rolled leaves later in the season are ten normal in external appearance.....

"Mode of Infection.- Hitherto infection of potato plants by F. oxysporum rough the root hairs and small rootlets has been accepted as the usual method. ith and Swingle said this occurred, and Manns reasserted the fact. In case of ie cowpeas and cotton, Orton found this manner of infection in both cases. omwell supposed root infection to be the means of entrance of Fusarium trachei-ilum Smith, causing the wilt disease of soybean. Jones and Gilman found the ots of cabbage to be attacked by Fusarium. These numerous instances would call r careful examination of the roots of infected plants. During the years 1916 d 1917, in only 6 plants out of many hundred examined was this method of infec-on determined as probable in the case of fusarium-blight of the potato in the eeley district. In 1918 the soil temperature at a depth of 6 inches was 6° F. ove the average for the month of June of the preceding two years. Plants of the harles Downing variety, planted during the last of May or early June, were badly iseased in some fields by being attacked through the fine roots and root hairs by . oxysporum. This one variety was more severely attacked than any other, even in ields containing several varieties. Most other varieties were not assailed in his manner at all, except a few scattering Early Ohio plants. Higher temperatures eem to be necessary for root infection.

"Infection from seed tubers containing the Fusarium organisms in the vascular undles has been very seldom found. Wollen-weber has shown that F. oxysporum verwinters in potato tubers, where it causes the familiar vascular discoloration. ith the sprouting of the eyes when the seed piece is planted the organism infects he new plants and presumably causes wilting and death. One of the most extensive-y advocated control measures has been aimed to avoid this kind of infection. No rouble has been experienced with this method of infection in the last three years. ield experiments tending to show the nonseverity of this method of infection will e given below.

"Seed-Piece Infection.- In the Greeley district and in other parts of Colorado otato seed pieces become infected with the Fusarium organism from the soil. The ut seed is vastly more liable to attack than the whole seed, and the decay follow-ng infection will begin two or three days after planting. It is justifiable to ssume that in the average field nearly all cut seed pieces are infected. Fields ave been examined in which hundreds of seed pieces were dug a few days after lanting, and less than 5 per cent were found to be free from infection. The in-ection occurs through the large open wound of the cut surface, lightly protected y callus. The interior loose parenchyma at the center of the tuber, farthest rom the active tissue of the vascular region, is the weakest and least protected. here is a difference in the susceptibility of seed of different varieties, but hat seems more important is that seed of the same variety from different sources aries greatly in its power of resistance. The rot following infection may be wift, and the fungus will destroy the seed piece before germination begins.....

Preventive Measures

"The use of whole seed is suggested, not as a means of controlling the blight, but of avoiding it. By the use of whole seed is meant not culls and other small potatoes, but tubers in good condition, well selected, and preferably of 1½ ounce weight or greater. Whole seed has been many times condemned as yielding quantities of unmarketable small potatoes, and from the horticultural point of view this is a serious fault. Under irrigation, however, the writer believes that whole seed can be made to yield nearly as many marketable tubers as cut seed. The increased stand resulting and the fact that no labor is required in cutting would promise a return commensurate with the initial increased cost of the seed. In one commercial field in 1918 the yield from whole seed was 100 per cent greater than that from cut seed of the same variety."- MacMillan, H. G. Fusarium-blight of Potatoes under Irrigation. Journal of Agricultural Research 16: 279-300. March 17, 1919.

References

(1) McKay, M. B. Control of potato diseases in Oregon. Ore. Agr. Col. Extension Bul. 186: 18. 1917.

(2) Manns, Thos. F. The Fusarium blight (wilt) and dry rot of the potato. Ohio Exp. Sta. Bul. 229. 1911.

(3) Orton, W. A. Potato wilt, leaf-roll and related diseases. U. S. D. A. Bul. 64. 1914.

(4) Bisby, G. R. Studies on Fusarium diseases of potatoes and truck crops in Minnesota. Minn. Exp. Sta. Bul. 181. 1919.

(5) MacMillan, H. G. Fusarium blight of potatoes under irrigation. Jour. Agr. Research 16: 279. Mar. 17, 1919.

(6) McKay, M. B. Potatoes diseases in Oregon and their control. Oregon Experiment Station Circular 24. February, 1922.

(7) _____ Transmission of some wilt diseases in seed potatoes. Jour. Agri. Res. 21: 821. Sept. 1, 1921.

(8) Goss, R. W. Potato wilt and stem-end rot caused by *Fusarium eumartii*. Neb. Exp. Sta. Res. Bul. 27, 1924.

(9) _____ Relation of environment and other factors to potato wilt caused by *Fusarium oxysporum*. Neb. Exp. Sta. Res. Bul. 23, 1923.

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* Note.- Recently Goss (8) has reported that potato wilt and stem-end rot in Nebraska are caused by *Fusarium eumartii* and not by *Fusarium oxysporum*, the species named at the head of this exercise.

DRY ROT OF POTATO

Caused by Fusarium coeruleum (Lib.) Sacc.

This is one of the worst, if not the worst, potato tuber rot in Oregon. The causal organism is closely related to the Fusarium wilt organism but confines its ravages to the tubers alone. Here it causes a dry rot of a part or all of the tuber. It is primarily a storage rot.

1. Symptoms.- Examine specimens. Note the sunken and wrinkled effect. Does any of the fungus show on the surface? Describe. Draw.

2. Life history.- This is a soil fungus and spores may get onto the tubers before or after digging and any wounding or bruising of tubers allows the fungus to enter. It develops in storage if the temperature is too high or the ventilation poor. It may be returned to the soil with diseased tubers. It is usually present, however, in soils of some localities, whether or not potatoes have been grown there.

Control

One of the most important features in control of tuber rots of this sort is that of proper storage. The following extract from Oregon Exp. Sta. Circular 24 briefly describes suitable storage conditions.

"Storage Losses Held Down by Low Temperatures and Good Ventilation.- A considerable part of the loss of potatoes due to disease is brought about by various tuber-rots in storage. There are a number of distinct rots of potato tubers caused by different organisms, and without exception they are most severe when the storage temperatures are high and when the air becomes heavily laden with moisture due to lack of proper ventilation. For instance, neither the powdery dry-rot which is so prevalent and serious in the eastern part of the State, nor the dry-rot which is apparently widely distributed all over the State, will develop seriously in storage if the temperature is kept from 35° to 40° F., and the storage place is kept well ventilated. It is also well known that the shrinkage in weight of potatoes in storage is much greater when the temperatures are allowed to remain high than when they are kept down to 35° to 40° F. These facts show the necessity of surrounding stored potatoes with the proper storage conditions in order that the serious losses from storage rots and from shrinkage in weight may be avoided.

"Potatoes in large quantities should never be placed directly in storage if they have become heated by high day temperatures. They should first be thoroughly cooled to prevent as far as possible the natural heating which takes place and to start the storage period under as favorable conditions as possible. This cooling may often be done with convenience and economy by leaving the potatoes outside of the storage place until well cooled by night temperatures and then moving them into storage either late at night or early in the morning before they have had an opportunity to become heated again by external conditions.

"Good Storage Places are Necessary for Trustworthy Storage.- A good storage place embodies the following requirements: ease and thoroughness of ventilation, ability to prevent rapid changes in temperature inside in response to rapid changes in the temperature of the atmosphere outside, convenience in cleaning, conveniently arranged for use, ample in size, and durable. In many sections of the United States where potatoes are an important cash crop these essentials are most efficiently secured in the cellars constructed mainly underground and partly above, with three or more ample-sized ventilators in the top, and with large doors and a driveway at one end large enough to accommodate a wagon for loading and unloading the potatoes.

"Another type of storage place used with much satisfaction in this State is the double-walled, well-insulated bin with the walls about six inches thick and with ventilators to let air in beneath the potatoes and out at the top; these ventilators, when the days are warm, may be left open at night and kept closed in the daytime, with the result that the whole bin is kept reasonably cool all the time. All storage places should be provided with a thermometer, so that the temperatures may be regulated intelligently. Injury is produced when the temperatures are too low as well as when they are too high. With the dry-rot it appears that the critical time, the time when many of the infections and when much of the loss from this disease results, is during the first two months after digging, when the temperatures are likely to be higher than during any other part of the storage period. It is at this time that our potatoes need especially to be surrounded by the favorable storage conditions that are best secured only in a good storage place. Whenever possible the storage place should be located in a dry, well-drained place, and preferably away from a sunny slope, so as to avoid unduly high temperatures.

"Bins Are Best Made up with False Slatted Walls and Floors.- An aid to thorough ventilation, the lack of which is now responsible for much of the poor storage in the State, all permanent potato storage places should be equipped with bins made of slatted floors, walls, and double slatted bin partitions. For convenience in taking down the bins when emptied of potatoes, these should be made in sections..... In any event the division wall and floor may be constructed of three-inch board strips, allowing a one-inch space between boards, and separated from the main floor and wall by joists and studding. This type of bin construction provides a conduit for air on the side wall, and, connecting with the bottom, affords an air passageway completely around the stored tubers, materially lessening the danger from tuber heating and from tuber-rots in storage and providing a convenient and sanitary bin which can be taken down easily, cleaned, and disinfected when desired.

"Bins Should be Cleaned and Disinfected Before Potatoes are Stored.- In order to kill all the rot-producing organisms already present, storage places, particularly those in which rotting of the potatoes was bad the previous year, should be thoroughly cleaned and disinfected before potatoes are again placed in storage. To accomplish this, the storage places may be swabbed or sprayed with either of the following solutions:

Copper sulfate (Bluestone) ..... 1 pound

Water .....:.....10 gallons

or

Formaldehyde ..... 1 pint

Water .....10 gallons

"After being disinfected the storage place should be well aired and dried out before potatoes are again placed in it."- McKay, M. B. Potato Diseases in Oregon and Their Control. Oregon Experiment Station Circular 24. pp. 18-20. February, 1922.

References

(1) McKay, M. B. Control of Potato diseases in Oregon. Ore. Agr. College, Ext. Bul. 186: 20. 1917.

(2) Carpenter, C. W. Some potato tuber-rots, caused by species of Fusarium. Journal Agr. Research. 5: 183. November 1, 1915.

(3) McKay, M. B. Potato diseases in Oregon and their Control. Ore. Exp. Sta. Circular 24. February, 1922.

POWDERY DRY ROT OF POTATO

Caused by Fusarium trichothecioides Wollenw.

Powdery dry rot is similar to dry rot of potatoes in some respects. Examine specimens, look at pictures and read descriptions of this disease. Compare it in all details with the dry rot. Write up carefully. Powdery dry rot prevails east of the Cascade Mts. in Oregon, while dry rot is common west of the Cascades.

References

(1) McKay, M. B. Control of potato diseases in Oregon. Ore. Agr. College, Ext. Bul. 186: 20. 1917.

(2) Orton, W. A. Powdery dry rot of potato. U. S. D. A. B. P I. Cir. 110. 1913.

(3) Pratt, O. A. Control of the powdery dry rot of Potato tubers caused by Fusarium trichotheciodes. Journal Agr. Research 6: 817. August 21, 1916.

Discussion

(See under "Dry rot" of potato.)

VERTICILLIUM WILT OF POTATO

Caused by Verticillium alboatrum Reinke and Bert.

This is the most prevalent and harmful wilt disease of potatoes in Oregon.

1. Symptoms.- These consist of a wilting of the plants and a discoloration of the vascular elements in both stem and tuber. The symptoms resemble closely those of Fusarium wilt. Here, too, the fungus may be present in the tubers without causing discoloration, as is true of Fusarium wilt. Examine and sketch.

2. Life history.- The causal fungus does not live indefinitely in the soil where no potatoes are grown as is true of Fusarium. It is transmitted through diseased tubers and by old diseased plants left in the field. It may spread from hill to hill in the field by growing from the roots of a diseased plant to the roots of the next plant in the row where the roots of the two plants mingle.

a. Conidiospores are formed abundantly on diseased stems and leaf parts. These do not appear to play an important part in the dissemination of the disease. Examine conidiophores and conidia. Draw.

Discussion.

The following extracts from two publications by McKay (2,3) give information about both of the potato wilt diseases. The second publication quoted, however, places especial emphasis on the Verticillium wilt.

"There are two serious wilt diseases in the State caused by distinct organisms, but the two diseases are so similar in appearance and effect that they may both be treated together. Attacked plants may wilt and die comparatively suddenly or they may succumb very gradually, finally dying only a week or two in advance of the unattacked plants in the same field. If the stalk of a wilted plant is cut across near the lower end, the interior will be found in most cases extensively browned, whereas in unattacked plants the tissue is normally white. If a thin slice is cut across the stem end of potatoes produced in a wilted hill, a distinct yellow to brown or black discolored ring, quite different from the normal appearance of the vascular ring which occurs as a faint layer located about $\frac{1}{4}$ inch beneath the skin, will generally be found present extending into the tuber and following the vascular ring for varying distances depending on the severity of the disease. Such discolored tissues indicate the presence in the interior of the potato of the organism that caused the wilting, though not all discolored tubers are carriers of the wilt fungus. Out of over twelve thousand tubers from wilted and healthy hills tested in detail during the past few years at Corvallis, only 45 per cent of the distinctly browned tubers were as a matter of fact invaded by a wilt fungus, while the other 55 per cent were apparently free of any wilt-producing organisms, though to all appearances the tubers could not be distinguished the one from the other. On the other hand, not all tubers which are invaded by a wilt fungus are discolored; 5 per cent of all the tubers examined and having no discoloration were really infected by a wilt-producing organism. This emphasizes the unreliability of depending on a bin examination of the tubers for the determination of the presence of wilt infection in seed potatoes.

"Careful Seed Selection Necessary to Avoid Wilt Infection in Tubers.- On the verage only about half of the tubers in a wilted hill are invaded by the organism hich caused the wilting. These, if used for seed, are very likely to give rise o the same disease the following year. Evidently the disease cannot be avoided ith any degree of certainty by cutting off and discarding the stem end and plant- ng only the eye end of affected tubers as has been formerly stated. This is due o the fact that the fungus responsible for the disease has apparently progressed arther into the tuber than the discoloration of the tissues would indicate. Then oo, as mentioned above, it is not possible to determine all tubers affected with ilt merely from their appearance.

"These facts just mentioned show that reliance for the control of wilt cannot ith confidence be placed on a mere bin sorting of the tubers for seed. Seed treat- ent is similarly not dependable for wilt control since this will kill only the rganisms on the exterior and is not effective in killing the fungus in the in- erior of the tubers. To avoid wilt in seed potatoes it is by far the best prac- ice to maintain a seed plot as outlined in a previous paragraph and rogue out all iseased plants as soon as they appear. In the case of wilt it is necessary to do his roguing more thoroughly than in the case of most other diseases. This is due o the fact that the disease has a tendency to spread from one plant to another in he row apparently by contact of the root systems. In one test conducted where the ilted plants were not removed during the growing season but were left in the field o the end of the season, thus giving the fungus an excellent opportunity to spread, he first, second, and third plants in the same row away from the original wilted lant carried on the average as high a percentage of wilt infection in the tubers s did the original wilted plant itself. In order to check this spread of wilt it eems advisable to pull out promptly not only the wilted hill but also the next ealthy hill on either side of the wilted one in the same row. By this method the eed plot can probably be freed of wilt in two years' time at most, whereas if only he wilted plants are pulled out, three or more years would undoubtedly be neces- ary to free the seed stock of this disease.

"Crop Rotation Must be Practiced to Avoid Wilt Infection From the Soil.- Since the organisms remain alive in the soil for a considerable length of time ollowing the production of a diseased crop, it is necessary to practice long otations in order to avoid undue infection from the soil. Even then absolute ontrol may not always be secured because one of the organisms, Fusarium, in some ases seems to appear more or less indiscriminately in some soils without any bvious relation to the crops grown thereon. This is apparently a minor source f the disease, however, and need not cause much loss where proper attention is aid to the use of the control measures already presented."- McKay, M. B. Potato iseases in Oregon and Their Control. Oregon Agricultural College Experiment tation Cir. 24: 26-34. Feb, 1922.

Transmission of Some Wilt Diseases in Seed Potatoes

"Among the diseases of the potato plant (Solanum tuberosum L.), the wilt diseases are the most widely known and are reported to cause the greatest loss the whole country over. There are several fungi which cause wilt of potato plants, but the most important and most frequently mentioned are Fusarium oxysporum Schlecht, and Verticillium albo-atrum Reinke and Berthold. These parasites have

een known for many years as a cause of disease in potatoes, and although many mportant facts concerning their nature and control have been learned by the ifferent investigators who have studied them, there still remain points of nterest and value concerning which there is a lack of adequate and definite nowledge and which will require much additional work to be completely cleared p. The investigations reported here were begun in order to secure some knowledge f the importance of these wilt diseases in Oregon and to determine the extent to hich the fungi causing them are transmitted from year to year in seed potatoes....

"This work was begun in 1916 and continued to the end of 1920. It has been conducted mainly in the vicinity of Corvallis, though field observations have shown that the organisms studied performed in very much the same way throughout western Oregon. However, the results secured probably could not apply in their entirety to the eastern part of the State where the soil, climatic and other conditions are very different. The work was begun with the supposition that Fusarium oxysporum was the organism chiefly responsible for potato-wilt in this region, but as the investigation has progressed it has been found that Verticillium albo-atrum is much the more serious of the two and that the former organism is relatively unimportant in western Oregon. Some of the tests begun for the purpose of determining the length of rotation necessary to avoid wilt infection from the soil can not be completed before a few more seasons of study. The work on the transmission of the organisms in seed potatoes has been terminated, however, and the results secured are given here.....

"Summary.- ..... In western Oregon, Verticillium alboatrum is more important than Fusarium oxysporum as a cause of wilt of potato plants. Each of these organisms is frequently present in the stem-end vascular region of potato tubers produced by diseased plants, and their presence there is usually though by no means always indicated by distinct discoloration of that region. An average of 6.6 per cent and occasionally as high as 14.4 per cent of the tubers which gave V. albo-atrum in culture were not discolored.

"Fusarium radicicola also is often present in this same region of the potato tuber, though it is not known to be a cause of wilt of the plants; and the discoloration generally accompanying it cannot be distinguished from that usually present with the two common wilt-producing organisms, Verticillium albo-atrum and F. oxysporum.

"Fusarium radicicola caused heavier discoloration than Verticillium albo-atrum in the infected tubers, while F. oxysporum caused still heavier discoloration than either of the other two organisms.

"In addition to these three fungi there are also several others that invade the stem-end vascular region of potato tubers, and the longer the potatoes are kept in storage the larger is the number of tubers which give organisms in culture. Some of these are parasitic on potatoes and many others are not, so far as known, associated in any way with a disease of this crop. They are usually though not always accompanied by discoloration of the tuber tissues.

"The presence of discoloration in the stem-end vascular region of potato tubers is not a trustworthy index of the presence of disease-producing organisms therein and ought not to be relied upon exclusively as a guide for the separation

of diseased from healthy tubers for planting purposes. For instance, 45 per cent of the tubers which were "browned" in the vascular region when cultures gave organisms which cause disease in potatoes, and 55 per cent gave either no organism or miscellaneous fungi of no apparent importance; 22 per cent and 5 per cent of those which were "yellowed" and "not discolored", respectively, gave organisms parasitic on potatoes, the others of these lots giving nothing of consequence.

"Verticillium albo-atrum occurred in all sizes of potato tubers, though somewhat more extensively in the small ones than in those of medium size. Fusarium oxysporum and F. radicicola did not seem to occur considerably more in one size of potato tuber than in another. The medium sized tubers gave a larger proportion of miscellaneous organisms than the smaller ones.

"Verticillium albo-atrum is transmitted to a considerable extent from seed potatoes to yields. From 30 to 50 per cent of the tubers grown from known infected seed potatoes were invaded by the same organism when harvested and cultured. Fusarium radicicola and F. oxysporum were transmitted to only a slight extent from seed potatoes to yields and were apparently not infrequently present in soils which had not been planted to potatoes for several years.

"The stem-end seed pieces did not seem to give more disease either in the plants or in the tubers produced therefrom than the eye-end seed pieces of the same infected tubers. The differences are at least so slight that it apparently is not advisable in attempting to avoid wilt in seed potatoes to rely much on the practice of discarding the stem ends and planting only the eye ends of tubers suspected of containing wilt organism.

"Verticillium albo-atrum is widely distributed in Oregon, having been recorded from 16 counties, and is the most important potato wilt-producing organism in the western part of the State. It causes appreciable losses each year, not only in commercial fields but especially in seed plots, because the presence of this fungus greatly lessens the value of the potatoes for seed purposes. It annually is responsible for the failure of a considerable number of fields to pass inspection for certification. The yields of affected plants are reduced on the average of from 30 to 50 per cent.

"Fusarium oxysprum is also widely distributed in the State, having been noted in all counties, though it is seemingly of little importance, at least in the western portion. In only a few cases have any considerable number of plants been found attacked by this organism in any western Oregon fields examined.

"Fusarium radicicola occurs frequently in potato tubers in Oregon, though it usually does not produce any apparent ill effects. It is often associated with a slight and generally rather dry, withered endrot or a soft and moist endrot, but it is not believed to be the sole cause of this trouble. It is more often found in tubers that are sound, except for having discolored vascular regions near the stem end.

"A stem-end rot of potato tubers, varying from a mere withering of the stem end to a dry, withered, brown to black rot, or to a soft jelly-like light brown rot, occurs rather commonly during some seasons in western Oregon. It appears to be due as much to climatic conditions during the growing season as to the presence

of any certain parasitic organisms."- McKay, M. B. Transmission of Some Wilt Diseases in Seed Potatoes. Journal of Agricultural Research. 21: 821-847. September 1, 1921.

References

(1) Orton, W. A. Potato wilt, leaf-roll and related diseases. U. S. D. A. Bulletin 64: 16. 1914.

(2) McKay, M. B. Control of potato diseases in Oregon. Oregon Extension Bulletin 186. page 18. 1917.

(3) McKay, M. B. Transmission of some wilt diseases in seed potatoes. Jour. Agr. Res. 21: 821-847. Sept. 1, 1921

PEACH SCAB

Caused by Cladosporium carpophilum Thum

1. Laboratory Study.

a. Symptoms.- Examine diseased specimens of fruits, twigs and leaves. Make drawings illustrating the symptoms on each and describe in notes.

b. Morphology of the causal fungus.- There is but one known spore stage in the life history of this pathogene, namely the conidial or imperfect stage. Examine sections through the disease lesions with the microscope. If sections are not available make your own mounts by scraping, with a scalpel, the surface of a fruit spot. Find and draw conidiophores and conidia. Be careful to distinguish the fungus from the hairs with which the surface of the peach is covered.

c. Notes.- Look up in the references a description of the symptoms, life cycle and control of peach scab and write full notes.

2. Text discussion.

Peach scab was first reported from Austria in 1877. Arthur (1) reported it in America in 1889. In 1917 Keitt (2) stated that the disease had been reported from 34 different states of the Union extending from the Atlantic to the Pacific Coast, and that serious losses may result from scab in practically all of the important peach-producing sections east of the Rocky Mountains.

Keitt (2) has given a detailed account of this disease and the following items are quoted from his bulletin:

"Description

"On fruit.- The earliest microscopic evidences of infection upon the green surfaces of the fruit appear as imperfectly defined, greenish to olivaceous, circular areas, usually less than half a millimeter in diameter. This effect is due to the appearance of the olivaceous conidiophores and conidia of the fungus among the hairs of the fruit. Upon blush surfaces, the early development of olivaceous color may be accompanied or preceded by the disappearance of the normal pink from the invaded areas, which remain much paler than normal until masked by the development of the fungus. In such cases a narrow peripheral zone of pale or yellowish green may mark the advance of each diseased area. The developing lesions gradually become larger, darker, and more clearly defined, owing to the death and disappearance of many of the obscuring hairs of the fruit and to growth and pigmentation of the fungus. When fully developed they are fairly well defined, circular, olivaceous to black areas with an average diameter of 2 to 3 millimeters, though under very favorable conditions they may attain a diameter of 4 or 5 millimeters. The direct injury is superficial, involving at most only a few layers of cells, which typically become separated from the adjacent normal tissues by the formation of cork. The infections occur practically exclusively upon the areas which are subject to thorough wetting. The lesions are, thus, generally most abundant about the peduncle and upon the upper portion of the wettable surface. They may be more or less uniformly scattered over the entire wettable surface, or they may be so abundant as to become confluent over large patches, frequently covering practically one-half of the surface of the fruit. The protected surfaces of even the most severly attacked fruits are usually free from infection. As the peaches grow rapidly just prior to maturity, the corked areas can not expand so readily as the normal tissues. This condition may result in mere inequalities of development between badly diseased and normal areas, but more commonly the growth stresses occasion the cracking of the cork layers. These cracks may appear upon individual lesions as small rifts extending slightly into the flesh, but upon very badly affected specimens they may extend the length of the fruit and penetrate to the pit. Badly scabbed fruits do not ripen evenly and are frequently inferior in flavor. Those which are severely scabbed about the attachment of the peduncle may be shed prematurely.

"On twigs.- On tender young green twigs the first macroscopic evidences of infection, typically, are barely visible, more or less imperfectly defined, very slightly raised, irregularly circular to oval areas, almost indistinguishable in color from the surrounding normal surfaces. As the lesions develop, their central areas become light brown, and later darker brown, and each is bordered by a slightly raised peripheral zone, usually about half a millimeter wide and of approximately the normal green color. The coloration and zonation, however, may vary considerably with such factors as the age and character of the twig, seasonal conditions, and the stage of development of the disease. At the end of the growing season, well-developed lesions appear as smooth, irregularly oval, light-brown to dark-brown areas of normal elevation, with slightly raised purplish to dark-brown borders, which usually vary from one-half to 1 millimeter in width and shade off peripherally into the color of the surrounding normal surface. Such lesions usually measure 3 to 5 by 5 to 8 millimeters, the greater development being parallel to the axes of the cells, the diseased areas becoming separated from the adjacent normal tissues by the formation of cork layers. Under favorable conditions, conidiophores, which bear typical conidia, may be produced upon the surfaces

of the infected areas at any time after the lesions become macroscopic. During the summer and fall following the infection, the conidiophores are ordinarily produced singly, and seldom in such quantity as to be macroscopically evident. In the following spring, however, they are produced abundantly in olivaceous tufts. These sporiferous tufts may develop over the entire surfaces of the lesions, or they may be confined to peripheral zones, usually 1 to 2 millimeters in width, leaving the grayish brown central areas bare. Such lesions may enlarge slightly during the spring and early summer of the second season. As the summer progresses and the bark is differentiated, however, they gradually begin to lose their identity. By the third summer, they usually become indistinguishable, while the fungus rarely survives the second winter. Lesions on water sprouts or very rapidly growing twigs may lose their identity with the rapid cork formation of the first summer. Twig infections are miscellaneously distributed upon the wood of the current year's growth. They may be sufficiently numerous to become confluent and form irregularly shaped lesions, which may attain a length of several centimeters. On account of their superficial nature, however, they rarely cause appreciable injury to the twigs.

"On leaves.- The first macroscopic evidences of infection appear upon the lower surfaces of the leaves as indistinct, imperfectly defined, some-what angular to circular discolored areas, usually one-half to 1 millimeter in diameter. The color of the affected surface may be pale green, almost indistinguishable from the normal, or it may vary to light brown or pinkish purple. The true color, however, is frequently modified or masked by an abundant development of olivaceous conidiophores and conidia. These are usually present in greater or less numbers when the infection first becomes macroscopic. When conditions especially favor their development, they may be borne in sufficient quantities to give a dark-green color to the lesions and to constitute their most conspicuous character. On the leaf lamina the fully developed lesions are somewhat angular to irregularly circular. Their average diameter is 1 to 2 millimeters, though in rare cases they may attain a diameter of 5 to 6 millimeters. On the petioles and midribs they are usually much longer and narrower, generally measuring 3 to 8 by one-half to 1 millimeters. The color of the blotch proper may vary from light brown to dark brown, or it may be pinkish purple. The demarcation remains indistinct and the elevation is usually unchanged, except in extreme cases, when the lamina above the larger lesions may become convex below and correspondingly concave above. When the lesions first appear, no evidence of disease is visible upon the upper surfaces of the leaves. In later stages, however, the tissues above and immediately around the affected areas may lose some or all of their green color and become pale yellow or purplish. In extreme cases such areas may die, but ordinarily no definite leaf spotting is induced. The lesions may occur miscellaneously scattered over the lower surfaces of the leaves and upon the petioles, or they may develop in such abundance as to become confluent and form irregular patches. The infection appears to occur with equal readiness on the petioles, midribs, veins, and leaf lamina. It was demonstrated in the inoculation experiments that badly diseased leaves may turn yellow and fall, but the writer has rarely observed this condition as the result of natural scab infection.....

"Morphology.

"Mycelium.- The morphological characters of the mycelium vary much with conditions. The very young hyphae are delicate, hyaline, branched, and septate. As the fungus develops, however, the diameter of individual cells normally increases, transverse and, more rarely, longitudinal divisions occur, and the walls of the more exposed cells thicken and become olivaceous...

"Conidiophores.- The conidiophores are short, erect, more or less flexuous, one to several septate, rarely branched, olivaceous hyphae, distinctly enlarged at the base and often tapering irregularly toward the apex.....

"Conidia.- The conidia are borne acrogenously, either singly or in short chains which may be branched when conditions especially favor sporulation. The spores appear first as small, rounded hyaline projections from the apices of the conidiophores. These bodies rapidly enlarge, assume the ellipsoidal shape of the immature spore, and become adjointed by septa from their sporophores.....

"If divided, the spores tend to be slightly constricted in the middle. The cells are very similar in size and shape, except that the basal ones tend to be slightly thicker and the apical less obtuse. In nature, normal mature conidia are fairly uniform in size. When they are produced in chains, the mature apical spores are indistinguishable in size and shape from the basal.....

"Dissemination of Conidia.....

"The results of these and of other experiments show: (1) That the dry conidia of Cladosporium carpophilum are not normally abscised or abjected; (2) that in dry or humid atmosphere they remain persistently attacked to the parent fungus; (3) that in contact with water they promptly become detached; (4) that even after spores have been detached by wetting the twigs they are not readily disseminated by wind after a period of drying. Thus, the evidence strongly indicates that under orchard conditions wind may be expected to play only a minor role in the dissemination of conidia, while meteoric water appears to be peculiarly well adapted to the performance of this function.

"Time of Natural Infection.

"Of fruit.- .....Abundant infection usually begins to occur about five to seven weeks after the petals fall and may continue until the fruit matures, but on account of the long period of incubation of the fungus the later infections do not become macroscopically evident.

"Of twigs and leaves.- From the results of the inoculation work it is evident that twig and leaf infection may occur whenever viable spores come in contract with tender young twigs or the under surfaces of leaves under conditions favorable for the development of the fungus.

"Overwintering of the Fungus.

"In considering the problem of how the fungus passes the winter, three major possibilities demand attention, viz. (1) the overwintering of conidia, (2) the overwintering of the mycelium in dead infected parts, and (3) the overwintering of the mycelium in lesions upon the living host.

"Careful studies of infected fruits, twigs, and leaves in late winter uniformly failed to reveal the presence of viable conidia. Furthermore, the evidence furnished by the germination tests previously reported shows conclusively that when exposed under orchard conditions these conidia would not remain dormant and viable over winter.....

"As has been shown in preceding topics, the primarily important overwintering of the fungus is accomplished by the mycelium in lesions on living twigs. Here the organism passes the winter safely, and with the advent of favorable conditions in spring it produces conidia abundantly.

Control.

"Early varieties.- Spray with self-boiled lime-sulphur, 8-8-50, or wettable sulphur, 5 pounds in 50 gallons,..... about one month after the petals fall.

"Midseason varieties.- Spray as for early varieties, adding a similar application about three weeks later.

"Late varieties.- Spray as for midseason varieties, adding a third application about one month after the second."

References.

(1) Arthur, J. C. Spotting of peaches and cucumbers. Ind. Agr. Exp. Sta. Bul. 19. 1889.

(2) Keitt, G. W. Peach scab and its control. U. S. D. A. Bul. 395. 1917.

BEAN ANTHRACNOSE

Caused by Collectotrichum lindemuthianum (Sacc.& Magn.) B.&.C.

This is a very common and destructive bean disease in some sections of the eastern half of the United States. In recent years it has been found to a limited extent in Oregon, having undoubtedly been brought in on diseased seed.

1. Symptoms.- Examine diseased specimens and note the characteristic spots on pods and seeds. Draw and describe.

2. Life history.- Examine sections of diseased pods or seeds and note the conidiophores and conidia. Draw. The conidia are apparently not very long lived and the disease is perpetuated by means of the mycelium which lives over in the spotted bean seeds.

References

(1) Edgerton, C. W. The Bean Anthracnose. Louisiana Exp. Sta. Bul. 119. 1910.

(2) Garman, H. A bean disease introduced in diseased seeds. Kentucky Exp. Sta. Cir. 16. 1917.

(3) Whetzel, H. H. Bean Anthracnose Cornell Exp. Sta. Bul. 255. 1908.

(4) Muncie, J. H. Experiments on the control of bean Anthracnose and bean blight. Michigan Exp. Sta. Technical Bul. 38. 1917.

Review

Compare bean anthracnose with bean blight as to symptoms, causal organism and control measures.

CALIFORNIA PEACH BLIGHT, OR CORYNEUM BLIGHT.

Caused by Coryneum Beijerinckii Oudem

This disease is prevalent in the Pacific Coast region and has been especially severe in California. Great damage is also caused in Oregon. The disease has also been reported from some states east of the Mississippi.

1. Symptoms.

a. On Fruit.- Examine diseased fruits and note characteristics of the spots caused by the pathogene. Draw and describe.

b. On twigs.- Note the spotting of twigs, blighting of buds, small cankers formed and the gumming which accompanies these symptoms. Draw and describe.

c. On leaves.- Note spots and shot holes. Draw and describe.

2. Life history of Causal organism.- Find the conidial pustules on the diseased twigs. Make mounts of conidia or examine slides already prepared and note shape and other characteristics of the spores. Draw. The chief periods of infection seem to be in the fall after the rains set in and again in May and June. Fall infections occur on twigs and buds, while spring infections occur on fruit and leaves. Look up the details in the references given below.

References

(1) Duggar, B. M. Fungous diseases of plants, p. 336.

(2) Hesler & Whetzel. Manual of fruit diseases, p. 311.

(3) Smith, R. E. California peach blight. Calif. Exp. Sta. Bul. 191: 73-98. 1907.

(4) Jackson, H. S. Oregon Exp. Sta. Crop Pest Report 1911-12, page 255.

(5) Cordley, A. B. & Cate, C. C. Spraying for peach spot. Ore. Exp. Sta. Bul. 106. 1909.

SEPTORIA DISEASES OF WHEAT

Caused by Septoria tritici Desm. and Septoria nodorum Berk.

Weber (1) describes two diseases of wheat caused by species of Septoria, namely, Speckled Leaf Blotch, caused by Septoria tritici and Glume Blotch, caused by Septoria nodorum.

These diseases have never been considered of great economic importance, but a few cases of considerable damage have been reported.

Laboratory.

a. Note the symptoms and make habit sketches.

b. Find the pycnidia on the dead spots. Examine sections or scrape of fragments of the pycnidia and mount. Make drawings. Compare with celery Septoria.

References.

(1) Weber, G. F. Septoria diseases of wheat. Phytopathology 12: 537. Dec., 1922.

LATE BLIGHT OF CELERY

Caused by Septoria petroselini Desm., var. apii Br. & Cav.

In regions where celery is grown this leaf spot disease sometimes causes serious damage.

1. Symptoms.- Note the spotes on the leaves and find the minute fruiting-bodies (pycnidia) of the fungus. Make habit sketch.

2. Causal organism.- Examine prepared sections of the pycnidia. Note shape and structure of the pycnidium as well as of the spores contained in it. Draw.

References

(1) Duggar, B. M. Fungous Diseases of Plants, p. 361.

(2) Duggar, B. M. Late blight of celery. Cornell Exp. Sta. Bul. 132: 206. 1897.

(3) Beach, S. A. Celery septoria. N. Y. Exp. Sta. Bul. 51: 137. 1893.

(4) Rogers, S. S. The Late Blight of Celery. California Exp. Sta. Bul. 208. 1911.

(5) Coons & Levin. Celery Blight. Mich. Exp. Sta. Special Bul. 77. 1916.

APPLE BLOTCH.

Caused by Phyllosticta solitaria E. & E.

Laboratory study.

a. Symptoms:- Examine fruit, twigs and leaves showing the characteristic symptoms of blotch. Sketch and describe in notes.

b. Morphology and life history.

(1) Pycnidia.- Only one stage in the life history of the fungus is known, namely the conidial or imperfect stage. With hand lens or dissecting microscope find the small black pycnidia. Do they occur on fruit, twigs and leaves? If not, where do you find them? Make an enlarged drawing of a lesion to show pycnidia.

Examine under a microscope a section thru a pycnidium. Note the relation to host tissue. Find spores within the pycnidium or escaped. Make a careful high power drawing of the section showing host tissue, pycnidium and spores. Describe in notes.

c. Read the following discussion quoted from Roberts (2) and from Gardner, Green and Baker (7), and write a complete account of this disease. Additional references are appended.

Text discussion

Historical

"In 1902 Clinton (8) published a description of the disease as it occurs on the fruit of the apple and considered it due to an unknown species of Phyllosticta. He also noted that pycnidia on the fruit produced fewer spores as the season advanced.

"In 1907 Scott and Quaintance (9) described the disease and gave tentative recommendations for its control.

"The first extensive report on the life history of the fungus and the first definite recommendations for its control were published by Scott and Rorer (3) in 1907. Later (1909), by comparison with type specimens they identified the fungus definitely as Phyllosticta solitaria E. and E. and determined by cross inoculations that the fungus found on leaves, fruit, and twigs is identical. They further determined that the fungus passes from one season to another in small cankers on the twigs.

"Sheldon (4) in 1907 reported the presence of the fungus on twigs. He was the first to identify the fungus as Phyllosticta solitaria E. and E. and gave a good description of the disease on leaves, fruit, and twigs.

"Lewis (1) gave a detailed description of the disease on the fruits of different varieties and noted particularly the destruction of fruit spurs."- Roberts, John W. Apple blotch and its control. U. S. Dept. of Agr. Bul. No. 534. June 28, 1917.

The following topics are quoted from Gardner, Green and Baker (7).

Geographical Distribution.

"According to the records of the federal plant disease survey, blotch seems to be confined to the central and southern states of the eastern half of the United States including the area between New Jersey, Nebraska, Texas, and Georgia. It is primarily a disease of the southern portion of the apple belt but extends northward to central Ohio, Indiana and Illinois and occurs here and there in the northern parts of these states. About half of the apple-producing area of the country is infested with this disease.....

Losses.

"According to the records of the federal plant disease survey the disease causes the heaviest losses in Oklahoma, Arkansas, Mississippi, Alabama, Georgia, South Carolina, North Carolina, Tennessee, and Kentucky, in which states the loss estimates for 1920 ranged between 5 and 10 per cent of the crop or an aggregate of 4,000,000 bushels. The same year the blotch loss estimates for the rest of the affected area aggregated 1,200,000 bushels.....

Varietal susceptibility.

"In Indiana the following varieties are severely attacked by blotch: Northwestern, Oldenburg (Duchess), Ben Davis, Smith Cider, Maiden Blush, Mann, Benoni, Akin, Willow, Gano, and Stark. For other parts of the country, the observations of previous investigators (1;2;3) and the collaborators of the federal plant disease survey would add the following to this list of very susceptible varieties: Missouri, Limbertwig, Wagener, Tolman, Lawver, Fameuse, and Lansingburg. The disease has been reported on many more varieties. In Indiana the cankers have also

been found on Rome, Transparent, Champion, Gideon, Rambo, Salome, Wealthy, Stayman, and Grimes and fruit lesions on York, Winter Maidon Blush, Winesap, Arkansas Black, Arkansas, Esopus, Stayman, Wealthy, Indiana Favorite, White Pippin, Jersey Black, Smokehouse, Gideon, Salome, McAfee, Kinnard, Jonathan, and Grimes.

"Possibly no varieties are absolutely immune to fruit infection but marked resistance is shown by the varieties, Grimes, Jonathan, York, Winesap, Delicious, and Stayman, all very popular in Indiana. From the commercial standpoint little trouble from blotch should be experienced with varieties not included in the list of those severely attacked of which Oldenburg is the most popular variety at present. Even the Rome and Transparent, while very subject to blotch canker, are not particularly subject to fruit infection. The blotch menace is real, however, in the case of Oldenburg and Ben Davis.

"The susceptibility of a variety to canker formation is a very important consideration because the persistence of the disease from year to year depends upon this factor. In Indiana, the Northwestern and Oldenburg varieties are highly subject to canker formation--much more so than Ben Davis and Stark. Cankers have been found only rarely on orchard trees of Grimes and Stayman and not at all on York, Jonathan, Arkansas, and Winesap.

"Cankers have been found on nursery stock of a number of varieties and on seedlings grown in the blotch regions. The wild crab-apple is also subject to blotch infection (4)."

"Effect of Blotch on the Fruit.

"The principal damage caused by this disease is due to the very objectionable and disfiguring fruit lesions. These are conspicuous shiney black blotches varying from one-eighth to one inch in diameter, irregularly lobed about the margin and often presenting a pattern composed of radiating outgrowths or projections. These blotches may be slightly elevated or distinctly sunken and bear near the center a group of very small black points or protuberances which are the flask-like spore-cases or pycnidia containing the spores of the blotch fungus. Very young fruit lesions may be small, slightly sunken spots, irregularly lobed and brown or tan-colored. Blotch lesions are most conspicuous on light colored varieties such as Northwestern, and are rather inconspicuous on dark colored varieties such as Arkansas and Lawver..

"Since the fungus grows more or less continuously within the tissues the size of the blotch depends somewhat upon the date of infection. The lesion is composed of a rather shallow superficial layer of tough black or brown tissue. The blotch fungus does not cause a rotting of the fruit, but in the case of very large lesions or of the coalescence of several lesions, cracks may be formed which permit infection of the underlying flesh by certain fruit-rotting fungi such as the black rot fungus or blue mold.

"Cankers on the branches.

"Young cankers first appear as dark or purplish raised spots varying in shape and bearing minute projecting points, the spore-cases or pycnidia of the blotch fungus. On twigs of older trees these lesions usually extend out from some part of the leaf base or leaf scar, but on suckers, watersprouts, seedlings, and nursery stock these may be located at or between the leaf scars.

"The center or older portion of the canker soon dries and becomes brown or tan-colored and slightly sunken, and is bordered by a crack or fissure, Each canker usually increases in size for several seasons by means of raised purplish marginal extensions which produce pycnidia and then dry out and become sunken and delimited by a marginal fissure like the central flake. More than one marginal extension may be formed during the second season. By this process combined with the coalescence of a number of cankers, large scurfy areas of bark may be produced

"On young actively growing Oldenburg and Transparent trees the cankers tend to encircle the branch, but seldom injure the cambium. Slow growing, shaded or suppressed twigs on old trees, however, may be girdled and killed by blotch infection, particularly on Oldenburg and Northwestern. Lewis (1) found that in the Missouri variety in Kansas, 28 to 42 per cent of the fruit spurs and 45 to 75 per cent of the buds on the remaining live spurs on the same branches were killed by blotch. The tendency of the cankers to occur at leaf scars results in the death of many buds.

"Blotch on the leaves.

"There are two distinct types of blotch infection on apple leaves as was first noted by Sheldon (4) in 1907. On the leaf blade tissue between the veins blotch produces very small white pin-head spots each of which bears a minute black dot in the center, the spore-case or pycnidium of the fungus. On the veins of the back of the leaf and on the petiole or leaf stem are produced elliptical or elongated, sharply sunken, light tan or buff-colored lesions, each bearing a number of the small black pycnidia of the fungus.

"Leaf infection is very heavy on the lower limbs of unsprayed trees and, while the white pin-head spots are rather harmless, the vein and petiole lesions must interfere to some extent with the translocation of the food materials and heavy infection near the base of the petiole causes many leaves to fall prematurely. In Kansas, Lewis (1) has noted severe defoliation in late summer as a result of petiole infection.

Life History.

"Blotch is caused by a parasitic mold or fungus (Phyllosticta solitaria E. and E.) which by its growth within the tissues of the leaf, twig, or fruit produces the spots or lesions characteristic of the disease..... Just under the skin on the lesions are formed the small flask-shaped spore-cases or pycnidia which are packed with very small colorless one-celled spores or "seeds" of the fungus.

"When mature pycnidia are wet with dew or rain, the closely packed jelly-like mass of spores inside absorbs water, swells rapidly, and oozes or forces itself out through a hole in the top of the pycnidium. During rains this extruded sticky mass of spores "dissolves" readily in water and the spores are separated and may be washed or splashed and blown in water drops to other parts of the tree.

"If one of these blotch spores lodges on the surface of a young leaf or fruit, it will germinate in a drop of rain or dew in a few hours by sending out an infection tube which will enter the host tissue. Here the tube grows and branches and becomes the feeding mycelium of the fungus which in a few weeks produces a visible blotch lesion. Within this lesion pycnidia and spores are soon produced, the latter to escape and spread the disease further.

"Whether the germ tube penetrates the host epidermis directly or through stomata or breathing pores has not been determined. Roberts(2) found that the fruit becomes resistant as it grows older but the occurrence of numerous small lesions on large fruit indicates that the fruit of some varieties may remain susceptible for a considerable period. The effective prevention of infection with the sprays applied at petal-fall and two, four, and six weeks thereafter is strong circumstantial evidence that most of the blotch infection occurs during a period of seven or eight weeks immediately after petal-fall. Anderson in Illinois reports some infection occurring in late August in 1921. Pycnidia bearing mature spores can be found at all seasons so it is very likely that spores of the fungus are spread during every rain between petal-fall and autumn and that the infection period is probably determined, as Roberts (2) has suggested, by the time during which the leaves and fruit are in a susceptible condition. The exact date when infection begins is of extreme importance since the spray program must be based upon it. In 1919, 1920, and 1921 infection started later than two weeks after petal-fall, but in 1922 it began somewhat sooner in Indiana and also in southern Pennsylvania, according to Walton and Orton (5).

"Cankers are the source of infection.

"Orchardists should thoroughly understand the significance of the canker stage of this disease because the cankers harbor the fungus over winter and are the source of infection for the fruit and leaves in the spring. The importance of the cankers in this respect was discovered by Sheldon (16) and by Scott and Rorer (6) as early as 1907.

"Scott and Rorer (3) found that the blotch fungus might live over winter in mummied fruits but did not produce spores. We have found in Indiana that the fungus also lives over winter in the petiole lesions on fallen leaves and viable spores have been found in such lesions early in the spring. However, such sources of early infection are considered unimportant.

"Pycnidia bearing mature spores are present in the twig cankers early in the spring and during warm rains spores ooze from these pycnidia and are washed and splashed to the new growth of leaves and fruit and cause infection. From the resulting lesions there may be a secondary spread of infection, but most of the infection probably comes from the cankers."

Control.

"Dormant sprays are ineffective.

"Dusts have given very poor control.

"Bordeaux sprays applied, 2, 4 and 6 weeks after petal-fall effectively prevent fruit, leaf and twig infection.

"Lime sulphur 1 to 40 is not as reliable as Bordeaux, particularly in canker control, but may be used to avoid russetting of the fruit.

"Bordeaux 2-4-50 has proved as effective as the 4-6-50 formula.....

"The sprays should be applied every year regardless of crop in order to prevent canker formation.

"Orchardists should reject infected nursery stock.

"Nurserymen should reject infected seedlings and should use bud sticks from sprayed or blotch-free trees. Nurserymen should apply the blotch sprays.

"In young plantings blotch should be eradicated by (1) spraying annually to prevent new cankers and (2) cutting out old cankers in early spring.

"Cankered branches and spurs should be pruned out.

"Cankers on the trunk and large limbs are shallow and can be shaved off with a sharp knife without injury to the underlying cambium. The cuts should extend half an inch or more beyond the visible margin of the canker. No disinfectant or wound dressing seems to be necessary. Healing occurs rapidly.

"Some cankers will be overlooked and a repetition of the operation the two following springs is essential."- Gardner, Max. W., Lauernz Greene, and Clarence E. Baker. Purdue Univ. Agr. Exper. Sta., Bulletin No. 267. January, 1923.

References.

(1) Lewis, D. E. The control of apple blotch. Kan. Agr. Exp. Sta. Bulletin. 196: 521-574. 1913.

(2) Roberts, John W. Apple blotch and its control. U. S. Dept. of Agr. Bul Bul. 534: 1-11. 1917.

(3) Scott, W. M. and Rorer, James B. Apple blotch, a serious disease of southern orchards. U. S. Dept. of Agr. Bur. Plant Industry Bul. 144: 1-23. 1909.

(4) Sheldon, J. L. Concerning the relationship of Phyllosticta solitaria to the fruit blotch of apples. Science 26: 183-185. 1907.

(5) Walton, R. C. and Orton, C. R. Time of apple blotch infection for 1922 in southern Pennsylvania. Abs. in Phytopath. 13: 11-12. 1923.

(6) Scott, W. M. and Rorer, James Birch. The relation of twig cankers to the Phyllosticta apple blotch. Proc. Benton County Hort. Soc. (Bentonville, Ark.) Aug. 8, 1907. p. 1-6.

(7) Gardner, Max W., Laurenz Green and Clarence E. Baker. Apple-blotch. Purdue Exp. Sta. Bul. 267. 1923.

(8) Clinton, G. P. Apple rots in Illinois. Ill. Agr. Exp. Sta. Bul. 69, p. 189-224, pl. A-J. 1902.

(9) Scott, W. M. and Quaintance, A. L. Spraying for apple disease and the codling moth in the Ozarks. U. S. Dept. Agr. Farmers' Bul. 283, 42 p. 7 fig. 1907.

CHAPTER XXII.

Diseases caused by parasitic seed plants.

Although the seed plants are not nearly as important as the fungi in regard to the number of parasitic species found in the group, yet there are several seed plants, belonging to the class Dicotyledonae, which do more or less damage as parasites upon other species of spermatophytes.

Parasitic seed plants may be classified as follows:

A. Stem parasites.
   1. Entirely dependent on host; ex. Dodder.
   2. Partially " " " ; " Mistletoe.

B. Root Parasites.
   1. Entirely dependent; ex. Broom Rape. (Orobanche)
   2. Partially " ; " Bastard Toad Flax. (Commandra)

DODDER OF CLOVER AND ALFALFA

Cuscuta sp.

Sometimes dodder attacks such plants as clover and alfalfa so vigorously as to smother out quite extensive areas.

1. Examine specimens showing the parasite twining around the host. Look for points of attachment to the host. Describe. Look for leaves and flowers on the dodder. Make a habit sketch.

2. Examine sections cut through host and parasite to show penetration of the parasitic roots (haustoria) of the parasite into the host plant. Note how the tissues and cells of the dodder connect up with corresponding tissues and cells of the host. Name these tissues and cells. Make a diagramatic drawing to show relationship of host to parasite in this respect.

References

(1) Hillman, F. H. Dodder in relation to farm seeds. U. S. Dept. Agr. Farmers' Bul. 306. 1907.

(2) Stewart, F. C. Troubles of alfalfa in New York. N. Y. (Geneva) Exp. Sta. Bul. 305: 355. 1908.

(3) Hansen, A. A. Dodder. U. S. D. A. Farmers' Bull. 1161. Jan., 1921.

(4) Robbins, W. W. and G. E. Egginton. Alfalfa dodder in Colorado. Colorado Exp. Sta. Bull. 248. November 1918.

Review

Describe the life history of a dodder plant. Where do seeds germinate? How do the seedlings find a host? Discuss the economic aspects of dodder and control measures.

OAK MISTLETOE

Phoradendron villosum Nutt.

1. Examine specimens of oak branches with mistletoe plants growing upon them. How are the parasites attached to the host as far as you can tell from external appearance? Draw.

2. Observe cross sections of oak branches cut so as to show the penetration of the mistletoe roots into the host. The roots, which clasp the stem and are more or less imbedded in the bark of the host, are known as bark-roots or cortex-roots, while those which penetrate into the wood of the host are known as "sinkers" or haustorial roots. Draw.

References

(1) Bray, W. L. The mistletoe pest of the southwest. U. S. D. A. Bureau Plant Industry. Bul. 166. 1910

(2) York, H. H. The anatomy and some of the biological aspects of the American Mistletoe. University of Texas. Bul. 120. 1909.

OTHER MISTLETOES

Razoumofskya spp.

Other species of mistletoe belonging to the genus, Razoumofskya attack various coniferous trees, such as pine, larch and fir. Examine specimens and compare with oak mistletoe.

References

(1) Wier, James R. Mistletoe injury to conifers in the Northwest. U. S. Dept. Agri. Bul. 360. 1916.

(2) Wier, James R. Larch mistletoe: Some economic considerations of its injurious effects. U. S. D. A. Bul. 317. 1916.

THE BROOM RAPES

Orobanche spp.

The Broom Rapes are typical dependent root parasites.

1. Observe dried specimen in flower, on herbarium sheet.

2. Observe younger specimen in museum jar. Note the bulbous base and the small root of a woody plant to which it is attached. Describe.

References

(1) Garman, H. The broom rapes. Kentucky Exp. Sta. Bul. 105. 1903.

(2) Muenscher, W. C. Orobanche ramosa on a Coleus. Rhodora 26: 133. July, 1924.

(3) Garman, H. The broom rape of hemp and tobacco. Kentucky Exp. Sta. Bul. 24. 1890.

BASTARD TOAD FLAX

Commandra umbellata Nutt.

This is an example of a partially dependent root parasite.

1. Observe dried plant in blossom, on herbarium sheet.

2. Examine specimens of cherry root with haustoria of Bastard Toad Flax attached. Note the slender yellow roots of the parasite with the swollen haustoria. Draw and describe.

References.

(1) Hedgcock, G. G. Parasitism of Commandra umbellata. Jour. Agr. Res. 5: 133. Oct. 18, 1915.

CHAPTER XXIII.

Diseases caused by parasitic animals.

There are many types of animal life, insects especially, which cause injury of one sort or another to plants, but there is only one group of animals that have been delegated to the plant pathologists rather than to the entomologists or zoologists for investigation. These animals are known as eelworms, nematodes or nemas.

There are three prominent types of injury by nematodes which we will consider here: (1) Root-knot, caused by Heterodera radicicola, (2) Leaf, bulb and stem-infesting nematode, Tylenchus dipsaci, and (3) Nematode disease of wheat, caused by Tylenchus tritici.

1. Laboratory.

a. Examine specimens of plants showing the three different types of disease mentioned above. Make drawings and describe symptoms in notes.

2. Read some of the following references and quotations and add any items of interest to your notes. Especially look up the life history of the worm and the methods of controlling it.

Discussion

The following quotation from Taubenhaus (6) gives a brief summary of symptoms, distribution, hosts and life history of the root-knot, or gall nema.

"Root Knot Caused by Heterodera radiciola (Greef) Mull.- Root knot is most prevalent in light soils. This, however, does not exclude it from heavier lands where it may sometimes be found. The trouble is most widespread in the Southern States, where the winter is mild. In unprotected places in the North its numbers are probably greatly reduced each winter. The annual financial losses from this disease are staggering in extent. With proper culture and fertilization, however, a crop may be produced with practically very little loss where neglect would have caused a total failure. This is especially true under greenhouse conditions.

"Symptoms.- The disease is characterized by a swelling on the roots, showing itself in small knots formed either singly or in pairs, or in strings, giving the affected root a beaded appearance. Sometimes, however, the swellings are so large that they may be mistaken for the root nodules of legume plants, which occur normally in great abundance. Infested plants usually linger for a long time, but they can be distinguished by a thin growth and yellow sickly looking leaves and stems.

"Distribution.- The eelworm seems to be of world-wide distribution, being found in Europe, Asia, Australia, and both North and South America. And yet, there are many localities in which this pest has never been known.

"Life History.- The eelworm is a very minute worm, seldom exceeding one twenty-fifth of an inch in length. It is semitransparent, so that it cannot be easily detected by the naked eye. In searching for the eelworm, break a fresh knot. Close examination will reveal two types of worms; a spindle-shaped worm, the male, and a pearly white pear-shaped organism, the female, firmly embedded in the gall tissue. The female is very prolific, depositing no less than 400 to 500 eggs during her lifetime. The eggs are whitish, semitransparent bean-shaped bodies, and too small to be noticed without the aid of a magnifying glass. The time which elapses until the eggs hatch depends largely upon weather conditions. In warm days the eggs hatch sooner than in cold days. Upon hatching, the young larvae either remain in the tissue of the host plant in which they have emerged, or, as is more often the case, leave the host and enter the soil. This is the only period during which the worms move about to any great extent in the soil, where they either remain for some length of time or immediately penetrate another root of the host. The nematodes in most cases become completely buried in the root tissue, establishing themselves in the soft cellular structure which is rich in food. The head of the worm is provided with a boring apparatus consisting of a sharply pointed spear, located in the mouth. This structure not only aids it in getting food but is also valuable in helping the young worms to batter through the cell walls before becoming definitely located.....

"Omnivorous Nature of the Eelworm.- There are almost five hundred species of plants known to suffer from the eelworm. This number includes all the important families of the flowering plants. According to Bessey the following are among the plants subject to root knot:

"a. Truck Crops.- Asparagus, bean, beet, cabbage, carrot, cauliflower, celery, chicory, cucumber, dill, eggplant, endive, gourd, Jerusalem artichoke, leek, lettuce, muskmelon, mustard, okra, onion, parsley, parsnip, pea, pepper, potato, pumpkin, radish, rutabaga, salsify, shallot, spanish oyster plant, spinach, squash, sweet potato, tomato, turnip, watermelon, yam.

"b. Garden Weeds.- Birdsfoot trefoil, burdock, carpetweed, dandelion, dead nettle, Florida beggarweed, horse nettle, lamb's quarters, mayweed, milkweed, nighshade, pigweed, plantain, pokeweed, ribgrass, shepherd's-purse, sheep sorrel, sow thistle, wild morning-glory."- Taubenhaus, J. J. Diseases of Truck Crops and Their Control. Pages 46-51.

There is also a species causing a serious disease of strawberries, clover and alfalfa and another causing a disease of wheat grains.

The Wheat Nematode.

Byars (3) gives the following description of the wheat nema.

"A serious disease of wheat known in Europe since 1743 and due to the nematode Tylenchus tritici (Steinbuch) Bastian has been recently found causing considerable damage in certain parts of the United States, particularly in Virginia.

"Reports in the literature and an examination of specimens received from various foreign countries show that the malady is almost world-wide in distribution, having been discovered in all continents except Africa. Within the United

States it is known to occur at present in California, Virginia, West Virginia, Maryland and Georgia, and other States also may be found to be infested.

"Samples of harvested wheat collected in Virginia contained from less than 1 percent to more than 50 percent of diseased kernels by count, and as much as 40 per cent damage by it to some fields of wheat in the same State has been observed.

"On seedlings the disease usually causes a decided wrinkling and rolling or distortion of the young leaves, which, if severely attacked, turn yellow, wilt and die. Occasionally such leaves have one or more small light-colored galls, which may be located on any portion of the leaf and contain the causal organism. Roots are not directly affected, nor do the stems of infected plants near the surface of the ground become swollen, as in the case of tulip-root, another disease of wheat, caused by the nematode Tylenchus dipsaci, which infects stems and crowns. It is on the maturing spikes that the malady produces the most conspicuous symptoms. As a rule, infected heads retain their green color longer than normal spikes. They are reduced in length and have their glumes spread out at greater angles than uninfected spikes. In place of normal kernels the glumes contain dark, hard galls, which are shorter and frequently thicker than wheat grains and which may be seen sometimes partly exposed in diseased spikes. Because of a general similarity, these galls often have been mistaken for the seed of cockle (Agrostemma githago), a weed which occurs commonly in wheat fields, for kernels affected by stinking smut due to Tilletia tritici, and for bin-burnt wheat caused by overheating the stored grain.

"The disease is caused by a minute nematode, Tylenchus tritici (Steinbuch) Bastian, the larval stage of which in great numbers may be seen with the unaided eye when a gall is teased apart in water. These larvae in mass appear milky white in color. Individually they are threadlike animals, nearly 1 millimeter in length and capable of an eel-like movement. Under favorable conditions of moisture and temperature they escape from the galls which have fallen to the ground or have been sown along with wheat seed, and by their own movement reach the young seedlings. They finally become located between the leaf sheaths near the terminal stem bud and in this region are passively elevated to the inflorescence. Occasionally they penetrate the leaves and induce small swellings, but usually they enter only the embryonic flowers, which develop into galls instead of kernels. In these places the larvae, after several metamorphoses, develop into about equal numbers of male and female adults, the former about 2 mm. in length and the latter from 3 to 5 mm. long. Each female may lay more than 2,000 eggs....., these eggs are elongate, ellipsoidal, granular bodies, which in a short time after oviposition develop into larvae and in this manner complete the life history of the organism. At the maturity of the plant the larvae within the gall become inactive, dried and motionless, in which lethargic condition they can remain alive for many years.

"The larvae inclosed in galls were killed by an immersion in water at temperatures of 50°, 52°, 54°, and 56° C. for 30, 20, 10 and 5 minutes, respectively, provided the galls were thoroughly moistened before the treatment. Much longer immersions at the same temperatures are necessary to kill them if the galls are dry when treated. Free from the protective gall the larvae succumb to temperatures

of 50° and 52° C. in 15 and 10 minutes, respectively; that is, in half the time required to kill them within the galls. Larvae either inside or outside the protective gall are highly resistant to the toxic action of formaldehyde, mercuric chlorid, copper sulphate, and sulphuric acid. They live after long submersion in concentrations of these chemicals, which in a short time are injurious to seed wheat.

"The nematode appears to be primarily a parasite of wheat. To a less extent it parasitizes tye, oats, spelt and emmer, and it has been reported on barley.

"The malady is most commonly spread by means of seed wheat containing nematode galls. It may be distributed in various other ways, such as by the feet of men and animals and by agricultural implements to which dirt containing viable larvae may cling. Surface water resulting from rains often transports the parasite from an infested to an uninfested field.

"The disease may be controlled by the use of nematode-free seed in combination with the employment of a 2 or 3 year crop rotation and the application of proper sanitary precautions. Uninfected wheat for planting should be secured, if possible, from localities where the disease does not occur. Nematode-free seed, however, may be obtained from diseased grain by floating off the galls in a 20 per cent salt solution and then treating the remaining wheat with water at a temperature of 50° to 52° C. for 10 minutes. Diseased seed can also be freed from nematodes without removing the galls by immersing them in water at a temperature of 54° to 56° C. for 10 to 12 minutes. By keeping wheat off infested land for two or better for three years, all or most of the nematodes will have starved, so that the subsequent wheat crop will not be appreciably damaged. Every precaution should be taken to prevent the reintroduction of nematodes to land which is being rotated to control the disease."- Byars, L. P. The Nematode Disease of Wheat Caused by Tylenchus tritici. U. S. Dept. of Agr. Bul. 842. pp. 35-37. September 7, 1920.

Leaf, stem and bulb nematode.

Tylenchus dipsaci Kuhn, or various strains of this species, is known to attack a wide range of host plants, among them being strawberry, clover, alfalfa, various bulbs as onion, hyacinth, narcissus, some weeds as false dandelion, and the wild strawberry. (For a further discussion of this type of nema see the following references below: 5, 7, 8).

References.

(1) Bessey, E. A. Root-knot and its control. U. S. D. A. B. P. I. Bul. 217. 1911.

(2) Byars, L. P. and W. W. Gilbert. Soil disinfection with hot water to control the root-knot nematode and parasitic soil fungi. U. S. D. A. Bul. 818. Jan. 5, 1920.

(3) Byars, L. P. The nematode disease of wheat caused by Tylenchus tritici. U. S. D. A. Bul. 842. Sept. 7, 1920.

(4) Fromme, F. D. The nematode disease of wheat in Virginia. Va. Exp. Sta. Bul. 222. August, 1919.

(5) McKay, M. B. A serious nematode disease of strawberry and clover in Oregon. Ore. Exp. Sta. Third Crop Pest and Hort. Rep't. 1915-20: 139-144. 1921.

(6) Taubenhaus, J. J. Diseases of Truck Crops and their control. 1918.

(7) Godfrey, G. H. The eelworm disease; a menace to alfalfa in America. U. S. D. A. Circular 297. 1923.

(8) Godfrey, G. H. and M. B, McKay. The stem nematode, Tylenchus dipsaci on wild hosts in the northwest. U. S. D. A. Bul. 1229. 1924.

CHAPTER XXIV.

"Virus" diseases.

Besides that group of diseases which are definitely known to be non-parasitic in origin and which were formerly quite generally referred to as the "physiological" diseases, there is another class which resembles the physiological diseases in some respects but which in other respects resembles the parasitic diseases. Here belong such diseases as mosaic and peach yellows. The latter has been referred to in the literature for over a century and a half. Mosaic as such has been known for a much shorter time. Most of the writings referring to mosaic have appeared during the last ten or fifteen years.

During recent years, since diseases of the mosaic type have been attracting more and more attention from plant pathologists, there has been a rapid segregation of virus troubles into many different types or phases of the malady. Ten years ago there were only three types of trouble in potatoes that were generally mentioned in discussing what was sometimes spoken of as the degeneration diseases in potatoes. These were mosaic, leaf-roll and curly-dwarf. Since then different investigators have described a large number of symptoms in potatoes which are all supposed to be manifestations of different types of virus diseases. Some of these supposedly different virus troubles that have gotten into the literature during this time, in addition to mosaic, leaf-roll and curly-dwarf are: Yellow dwarf, spindling-sprout, spindling-tuber, witches' broom, streak, unmottled curly dwarf, mild mosaic, leaf-rolling mosaic and rugose mosaic. On peaches three troubles have been recognized: Yellows, little peach and rosette. On bramble fruits the following have been named: mosaic, leaf-curl and streak or eastern blue-stem. The above are some of the different symptom-complexes that investigators have been able to distinguish. There is no assurance that still others will not be brought to light by more intensive work in the future. On the other hand it is possible that further research may show that some of the troubles already named are not separate and distinct types after all.

Symptoms.

The symptoms of mosaic as originally defined consist of a mottled appearance of the foliage due to difference in intensity of the green color, making a mosaic-like pattern of alternating dark green and paler yellowish-green spots or blotches on the leaves. More recently modifications of these symptoms have been observed. Some of these modifications have given rise to such names as mild mosaic, leaf-rolling mosaic and rugose mosaic. Detailed symptoms of particular virus diseases on specific hosts will be described later when these diseases are taken up in detail.

Cause.

The problem of determining the exact cause of these so-called "virus" diseases has been a baffling one. It has long been known that they can be transmitted from

one plant to another in various ways but all of them involving the transfer of some infective principle which seems to be carried in the plant juices. The disease has been transmitted by expressing the juice from a diseased plant and inoculating it into a healthy plant by needle punctures or better by mutilating the leaf of a healthy plant and then applying the disease-carrying juice. Also in many cases grafting operations have successfully transmitted the disease. In nature some of these diseases are carried by insects, notably aphids. The disease is transmitted in vegetative parts, in plants which are propagated by that means, such as potatoes and plants with perennial roots or underground stems. In a few cases it is said to be carried in seeds, notably in beans(14) and wild cucumber(15).

The fact that the diseases can be so readily transmitted has naturally lead to the supposition that an organism must be responsible for the trouble. However, the continued failure to find any organism, either with the highest powered microscope or by any cultural methods known, has led many to doubt that it is a trouble of parasitic nature, or at least that it is caused by any common bacterial or fungous organism such as cause all our well known parasitic diseases. Thus, due to its infective nature and to inability to find the causal agent, this type of disease has come to be known as a "virus disease."

Theories as to Cause.

There have been several different theories advanced as to the cause of mosaic and the other "virus" diseases, namely: (a) that the cause is an organism, an ultramicroscopic bacterium; (recently the claim was brought forward that protozoa are responsible); (b) that an enzyme is the cause; (c) that a "virus", which is neither an enzyme nor an organism, in the ordinary sense of the word, is responsible for the trouble.

Bacteria versus enzymes.

Probably Allard (1) and Freiberg (2) are the most clear cut exponents of there two theories. Allard maintains that the disease must be caused by an ultramicroscopic parasite of some kind. Freiberg thinks there is every reason to believe that an enzyme of some sort is the cause of the trouble.

Protozoa.

During recent years certain investigators have been finding peculiar bodies, suggesting in greater or less degree protozoa, in the cells of plants affected with mosaic. Kunkel (9) described such bodies for corn mosaic. The summary from his paper is quoted below:

"(1) A foreign body believed to be a living organism is invariably present in diseased cells of mosaic corn plants.

(2) The body is irregular in shape and always occupies a position on or near the host cell nucleus. It usually shows a definite reticulate structure stains like protoplasm and is frequently vacuolate.

(3) The distribution of the intracellular bodies corresponds exactly with the distribution of the light green color in diseased leaf tissue. In the stalk, they are present in the cells of diseased tissues but absent from the cells of healthy tissues.

(4) It is suggested that the bodies of corn mosaic may be similar to those associated with certain virus diseases of man and animals.

(5) Corn mosaic is similar to, if not identical with, the yellow stripe disease of sugar cane.

(6) Nine varieties of sweet corn, two varieties of pop corn and fourteen varieties of field corn have been shown to be susceptible to the disease. Several varieties are somewhat resistant but no variety is known to be immune."

McKinney, Eckerson and Webb (10) have recently described intracellular bodies associated with wheat rosette and leaf mottling. Their discussion of the possible nature of these bodies follows:

"The studies made to date show clearly that the bodies in question are not artifacts and that their nature is such that they do not yield readily to definite interpretation. While it is possible that the bodies may be organisms, it is also possible that they are the result of the reaction of the host cells to the disease. A rather comprehensive study of the literature shows that there are several possibilities in connection with the latter interpretation, some of which are more plausible than others, but a considerable amount of comparative study must be made before these interpretations can be definitely accepted or rejected.

"While it is recognized that the intracellular bodies associated with wheat rosette and leaf mottling may be a stage of some definite parasite, it is also recognized, on the basis of the cytological studies made thus far, that the distribution of the intracellular bodies in the host tissue and their apparent parallel development with that of the host cells do not seem to conform exactly with the distribution and development of any plant parasite known.

"In general, the intracellular bodies in question resemble the cell inclusions of unknown nature which are associated with some of the virus diseases of animals. This resemblance is especially striking in connection with certain of the Negri and Guarnieri bodies which are associated with rabbies and smallpox, respectively."

Nelson (11) seems to be the only person who has come out unequivocally and called these bodies protozoa. He claims that protozoa exist in the sieve tubes of bean, clover and tomato affected with mosaic and in potato leaf-roll plants. The publication of Nelson's paper stimulated feverish activity on the part of plant pathologists in the further investigation of this perplexing problem and while, as stated above, various other workers have found bodies of one sort or another they are very conservative in making any claims as to the exact nature of these bodies.

Protozoa in human medicine.

The fact that certain diseases in human beings, as malaria and sleeping sickness are known to be caused by organisms of the protozoa type and that these germs in case of malaria are carried by mosquitoes has led plant pathologists to search in this direction for a possible cause of mosaic and the other virus troubles, since these, too, are insect transmitted. Certain other bodies associated with diseases in the animal kingdom were referred to above in the quotation from McKinney, Eckerson and Webb. The many resemblances of this sort between the virus diseases in plants and certain maladies in the animal kingdom have undoubtedly been responsible for the adoption of the term "virus diseases" to designate this type of disease in plants.

The "virus" theory.

Some investigators have given up hope that this problem will ever be solved on the basis of any of the theories above mentioned, that is, they are convinced that this type of disease is not caused by either bacteria or protozoa, or by enzymes.

Duggar and his co-workers have probably done more fundamental research on this problem during the last few years than anyone else. It has been known for some time that the infective principle, whatever it may be, will pass through certain filters but is held back by other filters. This opened up an avenue through which one might determine, at least, the approximate size of the infective particles and so perhaps gain some clue as to their nature. Duggar and Karrer (12), in an attempt to determine the size of these particles, used filters with various sized openings. By finding filters through which the virus particles would pass and others through which they would not pass they were finally able to find filters with openings just large enough to allow the particles to pass. Then by testing these filters with known substances they were able to determine the size of the mosaic virus particles. They found that filters which were just porous enough to let blood haemaglobin through would also allow the virus particles to pass. In this way they estimated that the infective particles of the virus causing tobacco mosaic are approximately 30 millionths, or 3 hundred-thousandths of a millimeter in diameter.

In a later paper Duggar and Armstrong (13) continued these studies and attempted to formulate some sort of a concept as to the nature of the infective principle. Their conception of these virus particles can best be conveyed by quoting a few lines verbatum from their paper:

"Taking into consideration all the facts, we cannot avoid the impression, tentatively, that the causal agency in mosaic disease may be, in any particular case, a sometime product of the host cell; not a simple product such as an enzyme, but a particle of chromatin or of some structure with a definite heredity, a gene perhaps, that has, so to speak, revolted from the shackles of coordination, and being endowed with a capacity to reproduce itself, continues to produce disturbance and "stimulation" in its path, but its path is only the living cell."

Hosts.

Virus diseases have been described on a great variety of hosts including potato, tomato, tobacco, cucumber, lettuce, bean, cabbage, mustard, turnip, spinach, clover, blackberry, raspberry, loganberry, peach, corn, sugar cane, and a large number of uncultivated plants. We shall select only a few of these for further study, namely the potato, the bramble fruits and the peach.

VIRUS DISEASES OF THE POTATO.

The chief facts about potato mosaic troubles as known up to 1920 are given by Folsom (7) in the following summary of Maine Exp. Sta. Bulletin 292:

"(1) Potato mosaic has been known in Europe for many years and in Maine since 1912. At present it is well established in this state. It is also well distributed over the United States.

"(2) The mosaic of tobacco, a plant related to the potato, has been thoroughly studied and apparently is quite similar to potato mosaic. However, there are important differences between the two maladies.

"(3) Potato mosaic has certain marked effects upon the appearance, physiology, and yield of potatoes.

"(4) It is transmitted by the tubers of infected plants, whether or not these plants have had time to show the usual symptoms before death. This transmission is not modified by seed treatment.

"(5) Its infectiousness has been proved by means of grafting and of inoculations with juice from diseased plants.

"(6) Aphids, or plant lice, have been shown to be important agents for natural transmission of mosaic from one potato plant to another.

"(7) Transmission has been attempted by means of flea beetles, Colorado potato beetles (or 'potato bugs'), seed-cutting knives, soil, and contact of seed pieces, leaves, stems, and roots, but has not yet been demonstrated with any of these agents.

"(8) The effects of the spread of potato mosaic to healthy plants apparently cannot be avoided by the selection of hills or tubers or by discarding parts of the seed tubers, unless plant lice are controlled.

"(9) The relation between this spread and differences between localities, varieties, fertilizers, and spray methods are being studied but have not been fully determined.

"(10) Such spread has been reduced by roguing seed plots, that is, by removing mosaic hills as fast as they appear, and absolutely prevented by thorough control of certain kinds of insects.

"(11) Potato mosaic is difficult to control but it is now possible to recommend measures that promise to be helpful." - Folsom, Donald. Potato Mosaic. Main Agricultural Experiment Station Bulletin 292, pp. 157-158. August, 1920.

Schultz and Folsom have continued their work on the virus diseases of potato and in 1923 published an article (6) in which they describe seven different types of this trouble in potatoes. Their descriptions of these types together with a summary of the chief facts they discovered about transmission, etc. follow:

"Mild Mosaic.

"The characteristic symptoms consist of slight dwarfing, distinct mottling, wrinkling, and some ruffling. As will be shown later, it is more easily transmitted than rugose mosaic and streak. The tuber symptoms are a general average reduction in size.

"Leaf-rolling Mosaic.

"...The writers here use the term "leaf-rolling mosaic" as designating a symptom complex that so far has been irreducible to simpler complexes, and that consists of slight dwarfing, diffused mottling, wrinkling, slight ruffling, and rolling of the upper leaves. It is different from mild mosaic in respect to the distinctness of the mottling, the presence of rolling, and the effects in combination with the spindling-tuber disease, and is similar to it in infectiousness. The tuber symptoms are a general average reduction in size. It is distinct from leaf roll.

"Rugose Mosaic.

"A type of Mosaic much more infectious than mild mosaic, as will be shown later, is further differentiated by distinct dwarfing, more chlorosis and more diffused mottling, a more rugose type of wrinkling, and a tendency to show brittleness, spotting, streaking, leaf drooping and premature death, especially when in combination with the spindling-tuber disease... The tuber symptoms are a marked reduction in size.

"Streak.

"Most of the streaking seen by the writers has been associated with rugose mosaic, but in at least one case the latter was absent so that streak may be considered as a distinct symptom complex. The current-season symptoms appear in a certain order after leaf-mutiliation inoculation, as streaking and spotting, burning, brittleness, leaf dropping, and premature death, with no mottling except for chlorotic spots a few hours previously in the places where streaking and spotting occur, and no wrinkling.

Symptoms following tuber perpetuation are extreme dwarfing, wrinkling, curling, rugosity, brittleness, leaf dropping, and premature death...The tuber symptoms are extreme reduction in size, with some darkening, near the eyes, that resembles small initial infection spots of late blight; Phytopthora infestans de By. Cracking or splitting of the tubers has been observed for streak.....

"Leaf Roll and Net Necrosis.

"Leaf roll..... is characterized chiefly by dwarfing, chlorosis, rolling, uprightness, and rigidity; burning also is common. Phloem necrosis is a microscopic symptom of great value for positive identification. Leaf roll is less easily transmitted than any other degeneration disease considered in this paper. The size of the tubers is reduced. .....

Spindling-tuber Disease.

"The spindling-tuber disease is characterized always by spindliness and upprightness, and often by a darker green color and slight rugosity. The tubers are abnormally spindling, spindle shaped, cylindrical, and supplied with conspicuous eyes, these symptoms varying somewhat with the variety. A lighter red color obtains in spindling-tuber Red Bliss Triumph tubers than in the healthy tubers of this variety. This disease is readily distinguished from the mosaic diseases by the absence of mottling, and from leaf roll and streak. It is about as easily transmitted as mild mosaic and leaf-rolling mosaic, and therefore less easily than rugose mosaic and streak, and more easily than leaf roll. It reduces the amount of tuber progeny, but because of the difference in shape it may not be readily apparent that the size is reduced below that of uninfected plants.

"The spindling-tuber disease of the potato has been recognized for many years by growers and others by various names, such as 'running out,' 'running along,' 'off shape,' 'Poor shape,' 'reversion' and 'senility.' Observations made by the writers on this potato trouble from 1917 to 1921, in connection with investigations on mosaic and leaf roll, indicated its infectious nature. If strains free from this malady were planted near stock with a high precentage of "run out" tubers, a considerable percentage of such tubers resulted in a few years. Elimination of "run out" tubers by selecting only the good, normal-shaped tubers at the time of planting did not produce stock free from such progeny....

"Unmottled curly dwarf.

"....It consists of pronounced dwarfing, spindliness, dark green color of the foliage early in the season, wrinkling, rugosity, slight ruffling, curling, some rolling, uprightness, brittleness, burning, somewhat premature death, and spindling, gnarled, and cracked tubers....."

"Summary.

"(1) Degeneration diseases of potatoes, in the absence of known identifying causes, are symptom complexes whose elementary unit symptoms can and should be

determined in the same standard variety or varieties and in the same environment.

(2) In the Green Mountain variety, several degeneration diseases have been distinguished and transmitted--namely, mild mosaic, leaf-rolling mosaic, rugose mosaic, streak, leaf roll, spindling-tuber disease, and unmottled curly dwarf.

(3) In Green Mountains, mild mosaic was not transmitted by contact except in stem and tuber grafts.

(4) A leaf-mutilation method of inoculation has certain advantages over other methods. In Green Mountains this method transmitted mild mosaic, with the effectiveness increased by insect-cage or greenhouse conditions as compared to open-field conditions, by inclosure within a damp chamber, and by repetition.

(5) In Green Mountains aphids (Macrosiphum solanifolii Ashmead) transmitted mild mosaic, both alone and in combination with leaf roll and with spindling tuber, while negative results were secured with flea beetles (Epitrix cucumeris Harris) and Colorado Potato beetles (Leptinotarsa decemlineata Say.).

(6) In Green Mountains, aphids from plants with a "curly-dwarf" combination apparently consisting of leaf-rolling mosaic and spindling tuber together, transmitted the curly-dwarf combination to part of a hill and spindling tuber alone to the other part, distinction being made between different tuber units of the second generation.

(7) Leaf-mutilation inoculation transmitted both rugose mosaic and streak readily in Green Mountains.

(8) Leaf roll was transmitted neither by contact except in grafts, nor by leaf-mutilation inoculation.

(9) Spindling-tuber disease is characterized, and proofs given to its being a degeneration disease, spreading in the field, being perpetuated by the tubers (shown in part by mechanical measurements), and being transmitted by tuber grafts, stem grafts, leaf mutilation, and aphids.

(10) Unmottled curly dwarf was transmitted by leaf-mutilation inoculation and by aphids.

(11) Combinations of symptoms exist that include more than one degeneration disease in the same plant. Aphids sometimes transmit only one disease from such a plant, but more often transmit the combination.

(12) In Irish Cobblers, leaf roll was transmitted by grafting and by aphids but not by leaf-mutilation inoculation, which is successful with all other degeneration diseases tested.

(13) In New White Hebrons in 1921, leaf-roll and net-necrosis percentages increased with the average weight of the tubers.

(14) Leaf-mutilation inoculation of Green Mountains in 1920 effected intervarietal transmission of rugose mosaic, a combination of leaf-rolling mosaic and spindling tuber, and unmottled curly dwarf, but was ineffective with mild mosaic and leaf roll. Comparison inoculations with aphids with four of the sources of inoculum gave similar results, transmitting rugose mosaic to Green Mountains from curly-dwarf plants in the Rural group.

(15) Leaf-mutilation inoculation of Green Mountains in 1921 effected intervarietal transmission of streak, rugose mosaic and streak together, rugose mosaic, leaf-rolling mosaic, and mild mosaic in insect cages (uncontrolled in the open field) but not leaf roll. With spindling tuber (uncontrolled in the open field) present in the progeny, leaf-rolling mosaic having been transmitted formed a curly dwarf combination. Rugose mosaic was thus transmitted from Carman No. 3 (Rural group) to Rural New Yorker, and from this and two seedling varieties to both Green Mountains and Irish Cobblers, but the symptom complexes were not identical in any two varieties.

(16) Streak did not appear the same before and after tuber perpetuation.

(17) Comparison inoculations with juice in capillary glass tubes were much less effective than leaf mutilation, with rugose mosaic and streak.

(18) Aphid inoculations of Green Mountains in 1921 effected intervarietal transmission of leaf-rolling mosaic from curly-dwarf plants in three varieties of the Rural group, of unmottled curly-dwarf and spindling tuber (separately) from Irish Cobblers, of rugose mosaic from one seedling variety, and of leaf-rolling mosaic and rugose mosaic together from two seedling varieties. Aphids also transmitted rugose mosaic to Irish Cobblers from two seedling varieties.

(19) Leaf-mutilation inoculations of Green Mountains in 1922 resulted in current-season symptoms of streak, rugose mosaic and streak combined and rugose mosaic with and without streaking. Comparison inoculations with mild mosaic, leaf roll, and spindling tuber gave no current-season symptoms in the open field. Comparison inoculations under insect cages gave current-season symptoms with mild mosaic and spindling tuber. Several different symptom complexes yielded only rugose mosaic as the current-season effect of inoculation sometimes with rugose mosaic somewhat masked in the original complex. Results with Green Mountains were somewhat duplicated in Irish Cobblers and Bliss Triumphs, but some varietal modification of symptoms apparently occurred in these and seedling varieties.

(20) In greenhouse inoculations, transmission of mild mosaic from Bliss Triumphs to Green Mountains was effected to some extent with juice in capillary glass tubes, but not with immersion of a split stem in diseased juice. It was effected more readily with leaf mutilation as the number of inoculated leaves was increased, and more readily with aphids as the number of individual insects was increased, being possible with one individual aphid.

(21) In greenhouse leaf-mutilation inoculations, rugose mosaic was transmitted from a seedling variety to Green Mountains and from Green Mountains to Irish Cobblers, but leaf roll was not transmitted from Irish Cobblers to Green Mountains.

(22) Interspecific inoculations with leaf mutilation and aphids indicate that tobacco mosaic is not identical with potato mild mosaic, that tomato is susceptible to both of these mosaics and also to potato rugose mosaic, and that nightshade (Solanum nigrum L.) is susceptible to potato mild mosaic. Raspberry mosaic seems harmless to potatoes.

(23) The various degeneration diseases of potatoes are different as to their economic importance resulting from their distribution and effect upon yield rate and quality.

(24) Natural transmission by insects contributes to the difficulty of the control problem.

(25) Perpetuation occurs in tubers and not in soil alone.

(26) About 10 days were required for the mild mosaic virus to diffuse from inoculated leaves to the tubers.

(27) Mosaic plants from the same seed tubers sometimes show different symptom complexes in different environments. Mottling is suppressed by southern regions and by higher temperatures. Dwarfing of the tubers, and therefore reduction of yield rate, was more pronounced in a southern region.

(28) Shading tended to increase mosaic mottling and decrease leaf rolling.

(29) In duplicate plots leaf roll and mosaic were contracted by healthy lots growing between rows of diseased lots, more in some regions than others.

(30) Selection of tubers without knowledge of the parent plants can not eliminate seed from diseased plants infected late the preceding season.

(31) The digging of selected healthy hills progressively later in the growing season was correlated with greater numbers of aphids and with greater amounts of disease in the progeny.

(32) Hill selection in fields containing diseased plants throughout the growing season is disappointing as a means of eliminating disease, but sometimes gives better results than using unselected stock from the same field.

(33) Proximity and a heavy aphid infestation increased the spread of mild mosaic, while sufficient isolation from diseased stocks reduced it so that a state of freedom from the disease was maintained. Isolation by 30 meters was insufficient, and over 400 meters was sufficient.

(34) Conditions that reduced aphid dispersal from diseased to healthy hills also reduced the amount of disease transmission.

(35) Potato degeneration is largely, and possibly is entirely, a result of the increase of, and injury by, certain degeneration diseases.

(36) The potato degeneration-disease problem is on the whole similar for all potato-growing regions, but is complex enough to vary somewhat from one region to another." Schultz, E.S. and Donald Folsom. Transmission, variation and control of certain degeneration diseases of Irish potatoes. Jour. Agr. Res. 25:43. July 14, 1923.

TOMATO MOSAIC

While tomato mosaic resembles potato mosaic in some respects, there are some apparent differences which are worthy of note. Some interesting points about tomato mosaic as summarized by Gardner and Kendrick (19) are quoted below:

"Mosaic is readily transmitted by inoculating a healthy plant with some of the juice of a mosaic plant.

"The disease is spread largely by insects, especially plant lice, and by certain cultural operations, such as pruning and transplanting.

"No indication of mosaic resistance have been noted in the numerous strains and varieties of tomatoes tested.

"Diseased plants do not recover.

"Mosaic does not persist in the soil or litter.

"Mosaic is not transmitted through the seed.

"Tomato mosaic is carried over winter by certain weeds which send up new shoots from deep rootstocks each spring.

"In Indiana the disease occurs on four of these weeds, horse nettle and three species of ground cherry, and has been experimentally transmitted from each of these to tomatoes.

"Two of these ground cherry species are extremely abundant and are the principal mosaic carriers.

"A considerable percentage of the ground cherry shoots in old tomato fields have come up showing mosaic the next year and the second year after the tomatoes.

"The disease may spread several hundred feet from these fields.

" Practically no mosaic has been found among these weeds except in the vicinity of tomatoes, past or present.

"Once introduced, mosaic will persist and spread year after year in the weeds. Since many new fields are used each year for tomatoes this reservoir of mosaic infection is increasing at an alarming rate.....

"A critical feature is the presence of ground cherries in and near plant-beds where they may serve as a source of early infection for the tomato plants.

"Abundant evidence of the plant-bed origin of mosaic has been obtained. The disease is transported far and wide with the transplants.....

"Mosaic Control Suggestions

"1. Do not use tomato transplants from plant-beds in which mosaic is present. Guard against spread during transplanting.

"2. Eradicate all horse nettles and ground cherries in and near greenhouses, plant-beds, and tomato fields early in the season.

"3. Keep tomato fields free of all Solanaceous weeds, annual or perennial.

"4. Keep plant-beds free of all weeds and tomatoes during the summer.

"5. Keep greenhouses free of volunteer tobacco and tomato plants and all related weeds.

"6. Control insect carriers by spraying or fumigation." - Gardner, Max. W. and James B. Kendrick. Tomato Mosaic. Purdue University Agricultural Experiment Station Bulletin 261. p. 2 May, 1922.

VIRUS DISEASES OF BRAMBLES.

Raspberries, blackberries and loganberries suffer severely from virus troubles. A disease of raspberries called yellows or leaf-curl has long been known. In 1922 Rankin and Hockey (26) described two diseases of red raspberries, namely mosaic and leaf-curl. In 1923 Wilcox (25) described a virus disease of black raspberry which he named eastern blue-stem. Zeller (24) in 1923 described three systemic diseases of brambles occurring in Oregon, namely, mosaic, leaf-curl, and bramble streak. Zeller's "streak" is the same as the "Eastern blue-stem" of Wilcox.

Zeller (24) describes the symptoms of these three diseases as follows:

"Symptoms of Mosaic.

"The most important result of mosaic diseases in brambles is the gradual decline of the plants, which yield sickly fruit and vegetative parts. In three to five years the plants are useless or perhaps dead. The first symptoms are the mottling of the leaves and usually a slightly lighter, yellowish green color of the whole plant. In many varieties of plants these symptoms are accompanied with dwarfing. The mottling of the leaves, caused by lighter and darker areas of green in the same leaf, varies in its prominence due to the thickness and texture of leaf of the affected variety and the weather conditions under which the leaves are formed.

"The mottling of the loganberry leaves is rather masked under usual conditions although at times very distinct mottling occurs.... Soon, however, when the darker or healthier portions of the leaf outgrow the lighter parts, a crinkly leaf is produced which is very characteristic. During the spring of 1923 these crinkled leaves have usually appeared near the base of the laterals, for the symptoms are produced during cool weather. Sometimes all the leaves of a cane are crinkly and

mottled, and sometimes such abnormal leaves are limited to one lateral. Seldom does the whole plant show these symptoms.

"In the diseased red respberries mottled leaves are usually seen on the new shoots produced during cool weather. These have larger splotches of dark green; but in leaves produced during warmer weather the mottling approaches a finer speckling and in hot weather the newly-formed leaves usually show no mosaic symptoms whatever.

"In the black raspberries the first symptom is the strikingly splotchy mottling of the leaves, especially on the sucker growth and new tips which appear during the cooler spring weather.... The leaves of fruiting canes are usually reduced in size and lack the lustre of healthy leaves. Many times the terminal bud and leaves of a cane or lateral may be stunted, thus forcing the development of laterals on the canes or several laterals from one bud. As the disease progresses year after year there is a tendency toward smaller leaves which in black and red respberries show narrowed leaflets often with deep incisions.....

"Symptoms of leaf curl.

"Leaf curl of red raspberries, like mosaic, is a systemic disorder and affected plants never recover. The leaves of affected plants have shortened stems, arched and crinkled leaflets, and either an abnormally dark green or light yellow color. The crinkled leaf-surface has an appearance as though the veins are too short for the amount of tissue between them. Usually the main vein of each leaflet is arched or curled downward as also are the leaf margins. For the most part, however, the dark green leaves observed in the West show crinkle due to arching of tissue between all of the small veins, but there is very little tendency for the mid veins and margins to curl downward. As the disease progresses the leaves become very small and crinkled; the canes are dwarfed, thick and stocky. The fruiting laterals are upright, dwarfed, and bear curled, heavy, dark green, small leaves. Flowers appear somewhat later than normally, and the fruit is inferior and dry. Although this disease is uncommon, it has been observed on Cuthbert and Marlboro red raspberries in Washington and Oregon.

"Symptoms of Bramble Streak.

"Bramble streak was recently decribed by R.B. Wilcox of the United States Bureau of Plant Industry, under the name of Eastern Blue Stem of the black raspberry. Bramble streak is of the same general type of diseases as the leaf curl of the red raspberries, but it also produces some symptoms like those of mosaic diseases..... This disease stunts the bushes and checks their growth. The leaves of diseased plants are curled due to an arching downward of the mid veins of the leaflets. This curling of the leaves is most pronounced toward the tips of the fruiting laterals which are shorter than on normal plants. After two or three seasons the leaves on the shortened laterals are crowded and curled, giving a rosette appearance. As in mosaic diseases, there is a uniform mottling of the leaves, which is shown in the accompanying illustration. In some districts in the eastern states there are discolored markings and spots on the stems. Dark blue streaks like broad pencil marks run lengthwise of the canes or there may be small spots of the same

color more or less crowded, giving the stems abnormally dark blue shades, or there may be no discoloration of the stems. Bramble streak is another systemic disease which is transmitted chiefly through diseased tips." - Zeller, S. M. Mosaic and other systemic diseases of brambles in Oregon. Ore. Exp. Sta. Cir. 49. 1923.

Dissemination.

The chief means of scattering these diseases known at present is through setting out diseased tips. The bramble fruits are propagated vegetatively and since any part of a diseased plant is apt to carry the virus, it follows that any parts of diseased plants that are used for starting new plantations are likely to carry the disease. The part which insects play in transmitting these diseases is not definitely known at present but it seems reasonable to suppose that since aphids transmit many of the known virus diseases, they probably are concerned with transmitting these bramble diseases also.

Control.

The two logical steps in control are naturally the roguing out of diseased plants and the selection of clean nursery stock, preferably from a neighborhood known to be entirely free from these troubles. If insects should be shown to carry these diseases then insect control will be a third item in the campaign against these virus troubles of the bramble fruits.

VIRUS DISEASES OF THE PEACH.

There are three different types of trouble affecting peaches which are now attributed to a "virus". These are peach yellows, little peach and peach rosette.

YELLOWS.

The following quotation from Hesler and Whetzel (27) gives the main facts as known about peach yellows:

"Wherever this disease occurs it is known as yellows, or peach yellows. It is primarily a trouble of the peach and nectarine, although it has been observed on almonds, apricots and Japanese plums. Seedling peaches are said to be more sensitive than budded trees. Seedling trees derived from budded fruit are equally susceptible.

"The origin of the trouble is unknown. Records of it date back to 1760, and in 1806 it was the subject of horticultural writings. The first discovery of peach-yellows is thought to have been made in Pennsylvania, near Philadelphia. Outbreaks of the disease occurred in the East in 1791, 1806, 1807, 1817, to 1921, 1845, to 1858, and in 1886 to 1888. It appeared in Michigan about 1869, or earlier. The disease is not known on any other continent than North America, and the early records coming from the eastern part of the United States would, therefore, seem to show that peach-yellows is native to eastern United States. It occurs as far south as southern Virginia, west to Arkansas and northeastern Texas, north to Canada, and east to New England.

"Peach-yellows is conceded to be one of the most serious diseases of the peach in America . It has been extremely ruinous in the region outlined in the preceding paragraph. Soon after its discovery, hundreds of orchards along the Atlantic Coast were destroyed by the disease, causing growers in many localities to abandon Peach-culture. In one county in Michigan the number of peach trees destroyed by yellows between the years 1874 and 1890 is estimated at more than one-half million.

"Symptoms.

"The marked symptoms of peach-yellows consist (1) in a premature ripening of the fruit; (2) a red spotting on the surface of the fruit; (3) the development of secondary shoots in great numbers, these being dwarfed and unhealthy in appearance; (4) the development of short shoot-axes with sickly foliage of a yellowish or reddish brown color, having a tendency to roll sidewise. These symptoms begin to develop in the middle of the summer. Subsequent developmental stages are observed at various seasons of the year....

"Cause of Yellows.

"Just what causes peach-yellows is still unknown. It therefore falls into a class, from the causal standpoint at least, with rosette and little-peach... It was long ago established that the disease is communicable, that is, the causal factor may be transferred from a diseased tree to a healthy one, and after a time the latter tree will show symptoms of yellows which in due time run their course. Buds from diseased trees convey the causal factor of yellows to the stock in which they are inserted. The inoculum, whatever its nature, is apparently carried in a few diseased cells which, if induced to unite with normal cells of the stock, will cause the stock to become diseased. Diseased seeds also carry the inoculum; and there are probably other ways in which it is carried.

"Control.

"The knowledge of remedial measures is about as meager as that of the causal relations. "Cures" are worthless and impossible.

"The prompt removal and destruction of affected trees has been recommended since 1828. The advice is still reliable. It has been practiced effectively in the State of Michigan in years past. The eradication of diseased trees must be an annual operation until no traces of yellows remain. Where no such efforts have been made to exterminate the disease, it has prevailed to such an extent that orchards have been destroyed and the culture of peaches abandoned....

LITTLE PEACH

Hesler and Whetzel (27) make the following statements about little peach:

"Like peach-yellows this disease is confined to the northeastern United States. It has been known for but a few years in this country. The origin of little-peach

is unknown, but the suggestion has been made that it came from abroad with Japanese plums. The first appearance of the disease in America was once believed to have been in Michigan prior to 1893, but there are some indications now that it appeared in New York State at an earlier date. More recently important notices of little-peach have come from New Jersey and from Ontario, Canada.

"Symptoms.

"Little-peach may or may not appear on a tree at the same time as yellows.....

"Summarizing the prominent symptoms of little-peach, the following should be noted: the fruit remains small, about one-half or one-third its usual diameter, it ripens about ten days later than normal fruit, and possesses a bitter flavor and a stringy flesh; the leaves are small, light to yellowish green, and droop or incurve to some extent.

"Cause.

"This phase of little-peach has been the subject of no little investigation, but practically no progress has been made. The cause is not known, but it is supposed that the factor here involved is closely allied to that of peach-yellows, but that the two diseases are distinct and entirely independent of each other. The disease is contagious; the causal factor can be carried by budding, although the disease may not come into evidence until the second year after inoculation in this fashion.

"Control

"So far as known, diseased trees have never been cured.... The only remedy known is the removal and burning of diseased trees...

PEACH ROSETTE

Rosette is briefly discussed by Hesler and Whetzel (27) as follows:

"The disease was first described as a probable southern variation of peach-yellows, but later it was referred to as a distinct disease. The earliest record dates back to 1879, when it was observed in the State of Georgia. Ten years later it appeared in Kansas. It has subsequently been reported from South Carolina, Arkansas and elsewhere.

"Symptoms.

"Peach-rosette is recognized by the development of a rosette or whorl of sprouts on affected limbs. The whole tree, or only one or two limbs, may be diseased. Within six to twenty-four months the tree dies. First evidence is noted in the early spring when the buds open. Instead of only a few winter buds pushing out, a great majority of them grow into shoot-axes in compact tufts or rosettes. Such are only two to three inches long, and they bear numerous small leaves. When older and larger leaves are affected, they show an inrolling of the

margins and a peculiar stiffening due to the midrib becoming straight. Smaller leaves are seldom rolled. Affected foliage turns yellow early in the summer and falls prematurely. Often leaves are blotched, browned and deadened at the ends and margins from the attacks of secondary leaf fungi. Fruit on rosette trees falls prematurely. Diseased trees show less tendency to develop sprouts on the trunk and main limbs.

"Peach-rosette differs from yellows in the more tufted character of the growths; in the absence of premature ripening of the fruit; more rapid destruction; and usually rosette is confined to the tips of branches, occurring but rarely on trunks and at the base of main limbs. It resembles yellows in the pushing out of dormant (adventitious) buds which develop diseased branches; the dormant buds tend to unfold in the summer and autumn; and only a portion of a tree may be affected, whereas the remainder is normal."

Cause.

The disease is probably of virus origin as are yellows and little-peach. McClintock (30) calls it an infectious mosaic disease. Parts of his summary are quoted below:

". .....rosette is readily transmitted from peach to peach by infected buds.

"Rosette has also been transmitted to two varieties of apricots, two varieties of cultivated plum, one wild plum, one cherry, and two varieties of almonds by means of infected buds.

"On some hosts rosette produces a mottling of the leaves similar to mosaic.

"Limited tests indicate that rosette is not soil-transmitted.

"Numerous attempts to transmit rosette by means of sap from diseased trees have proved unsuccessful.

"In a large number of transfers of various types of insects from rosetted trees to healthy susceptible hosts, not a single case of rosette was transmitted."

Control.

"The prevention of rosette is not known to be possible. All affected trees should be promptly destroyed. This should be done early in the spring as soon as the disease appears, and before the leaves fall. In those regions where the disease is present, attention should be given to the plum, which at times shows symptoms of rosette similar to those described for the peach."- Hesler and Whetzel, Manual of fruit diseases. (27).

References.

(1) Allard, H. A. Some properties of the mosaic disease of tobacco. Jour. Agr. Res. 6: 649. July 24, 1916.

(2) Freiberg, G. W. Studies in the mosaic diseases of plants. Ann. Missouri Bot. Gard. 4: 175. 1917.

(3) Taubenhaus, J. J. Diseases of Truck Crops. 1918. pp. 84-85.

(4) Schultz, E. S., et al. Investigations of the mosaic disease of the Irish potato. Jour. Agr. Res. 17: 247. Sept. 15, 1919.

(5) Schultz, E. S. and Donald Folsom. Transmission of the mosaic disease of Irish potatoes, Jour. Agr. Res. 19: 315. July 1, 1920.

(6) _____ _____ Transmission, variation and control of certain degeneration diseases of Irish potatoes. Jour. Agr. Res. 25: 43. July 14, 1923.

(7) Folsom, Donald. Potato mosaic. Maine Exp. Sta. Bul. 292. 1920.

(8) Orton, W. A. Potato wilt, leaf-roll and related diseases. U. S. D. A. Bul. 64. 1914.

(9) Kunkel, L. O. A possible causative agent for the mosaic disease of corn. Bul. of the Hawaiian Sugar Planters' Association, Vol. III. No. 1. 1921.

(10) McKinney, H. H., Sophia H. Eckerson and R. W. Webb. The intracellular bodies associated with the rosette disease and a mosaic-like leaf mottling of wheat. Jour. Agr. Res. 26: 605. Dec. 22, 1923.

(11) Nelson, Ray. The occurrence of protozoa in plants affected with mosaic and related diseases. Mich. Exp. Sta. Technical Bul. 58. 1922.

(12) Duggar, B. M. and Joanne L. Karrer. The size of the infective particles in the mosaic disease of tobacco. Ann. Mo. Bot. Gard. 8: 343-356. 1921.

(13) Duggar, B. M. and Joanne K. Armstrong. Indications respecting the nature of the infective particles in the mosaic disease of tobacco. Ann. Mo. Bot. Gard. 10: 191. Sept., 1923.

(14) Stewart, V. B. and Donald Reddick- Bean mosaic. Phytopath. 7: 61. Feb. 1917. (Abstract of paper read at 8th. annual meeting of the American Phytopathological Society at N. Y. City., Dec. 26-30, 1916.

(15) Doolittle, S. P. and W. W. Gilbert. Seed transmission of cucurbit mosaic by wild cucumber. Phytopath. 9: 326. Aug., 1919.

(16) Barrus, M. F. and C. C. Chupp. Yellow dwarf of potatoes. Phytopathology 12: 123-132. 1922.

(17) Elmer, O. H. Mosaic cross-inoculation and insect transmission studies. Science. n. s. 56: 370. 1922.

(18) Johnson, James. The relation of air temperature to the mosaic disease of potatoes and other plants. Phytopathology 12: 438. 1922.

(19) Gardner, Max. W. and J. B. Kendrick. Tomato mosaic. Purdue Exp. Sta. Bul. 261. 1922.

(20) Doolittle, S. P. The mosaic disease of cucurbits. U, S. D. A. Bul. 879. 1920.

(21) Goss, R. W. Effect of environment on potato degeneration diseases. Neb. Exp. Sta. Research Bul. 26, 1924.

(22) Brandes, E. W. Mosaic disease of corn. Jour. Agr. Res. 19: 517. Aug. 16, 1920.

(23) ..... Artificial and insect transmission of sugar-cane mosaic. Jour. Agr. Res. 19: 131. May 1, 1920.

(24) Zeller, S. M. Mosaic and other systemic diseases of brambles in Oregon. Ore. Exp. Sta. Cir. 49. 1923.

(25) Wilcox, R. B. Eastern blue-stem of the black raspberry. U. S. D. A. Cir. 227. 1923.

(26) Rankin, W. H. and J. F. Hockey. Mosaic and leaf curl (yellows) of the cultivated red raspberry. Phytopathology 12: 253. 1922.

(27) Hesler and Whetzel. Manual of Fruit diseases. 1917.

(28) Blake, Maurice A. Peach yellows and little-peach. N. J. Exp. Sta. Bul. 226. 1910.

(29) Blake, M. A., M. T. Cook and C. H. Connors. Recent studies on peach yellows and little peach. N. J. Exp. Sta. Bul. 356. 1921.

(30) McClintock, J. A. Peach rosette, an infectious mosaic. Jour. Agr. Res. 24: 307. Apr. 28, 1923.

CHAPTER XXV.

Non-parasitic diseases.

There are many disorders of plants which are of non-parasitic origin and are frequently spoken of as "physiological diseases". As a rule the causes of these diseases are obscure and very difficult to determine.

Among the so-called "physiological diseases" or non-parasitic diseases may be mentioned such troubles as Bitter Pit, Cork, Jonathan Spot and Water Core of Apple, Drouth Spot of Prune, Point Rot of tomato and Internal Browning and Black Heart of potato.

BITTER-PIT, OR STIPPEN OF APPLE

Probably caused by unbalanced water relations.

The following discussion is quoted from Hesler and Whetzel (9).

"Symptoms.- The fruit only is affected by stippen. The disease may be found toward the end of the growing-season, at least after the fruit is half grown; or it may not be seen until after the fruit has been placed in storage. Fruits approaching maturity seem to be in the most critical stage of their development so far as bitter-pit is concerned. Its appearance amongst the fruit on a tree is very erratic. Sometimes only one apple on a twig is affected. Again, all the fruits of a cluster are pitted. It has, beyond reasonable doubt, been demonstrated by means of the X-rays that the disease develops internally before there are any external signs of it. The first outward symptom of the disease is the appearance of slightly sunken spots here and there over the surface, usually most numerous toward the blossom-end. These spots or pits tend to a circular form and vary from mere dots to depressions measuring a quarter of an inch or more in diameter. They look like hail or sand bruises. On a red variety the color is at first darker red, while on a green variety the spots are darker green. Finally the depressed areas are brown. As the fruits mature the pits become more numerous and deeper, but the skin covering them is not broken nor ruptured in any way. Another form of the disease is found in what is known in the western United States as hollow-apple. This form is sometimes called confluent bitter-pit or crinkle because the upper surface of the fruit develops rough folds giving it a crinkled appearance.

"If an affected apple is cut open, an area of the flesh directly beneath a pit is found to be dead, brown, dry and spongy, or corky. On account of these characters various names have arisen: dry-rot, apple brown-spot, beige (cork disease) and others. Throughout the pulp of the apple there are usually found brown streaks or spots of dry, spongy flesh which do not extend to the surface of the apple, and which are not connected with the surface lesions. Where the pits and internal brown spots are numerous the apple may have a bitter taste, whence the name bitter-pit.

There are many appearances similar to, or mistaken for, bitter-pit. The apple is subject to several fruit-spot diseases, some of which may be regarded as identical with stippen by the casual observer. The New England fruit-spot is distinguished from stippen by its spots being decidedly less sunken, and by the lack of semblance to a bruise. In the fruit-spot one or more black pimples develop on the affected area; in the case of stippen there is no evidence of such structures.

"Bitter-pit or stippen is frequently confused with hail marks and bruises of various kinds. In the case of any such injuries, however, the skin has the appearance of having been pushed in rather than shrunken, and also mechanical injuries often show a broken skin, a feature not characteristic of stippen. Scab is distinguished as follows: It shows at first as a velvety, then as a corky lesion, not depressed. The various apple-rot lesions are not depressed until much larger than stippen-spots. San Jose scale marks are sometimes regarded as bitter-pit lesions, but the former does not show the plain depression of the latter. Most growers will not mistake other diseases for bitter-pit.

"Cause of stippen.- Stippen is a non-parasitic disease, one in which the primary cause is not certainly known, but one for which all parasitic organisms, such as bacteria and fungi, are in no way responsible. The common opinion of those who have given some consideration to this phase of the subject is that the pits result from a disturbed water relationship. As to the nature of this disturbance, however, authorities are not entirely agreed.

"Since every grower has his own opinion in regard to the cause of this disease it may be of interest to give some of the theories of those who have most carefully investigated it. In order to make these theories clear it is first necessary to have clearly in mind the general structure of the apple-fruit and the nature of the spots. The flesh of the growing apple is made up of rather large egg-shaped cells within which the living substance of the fruit is enclosed, together with the water and mineral substance brought up as food from the roots, and the starch made by the leaves and green fruit. As the fruit ripens the starch is converted into sugar. Passing from the twig into the fruit, by way of the fruit stem, are large numbers of sap-tubes which, as they enter the base of the fruit, separate, and branching permeate the flesh in all directions. These sap-tubes are the channels through which water and mineral food from the soil are carried to the living cells of the fruit. The apple is enclosed in a smooth, water-proof skin made up of a layer of cells, the outer walls of which are thick with a waxy infiltration of water-proof nature. Scattered here and there, but most abundant toward the blossom-end of the apple, are minute openings through the skin known as lenticels. They are visible to the naked eye on certain varieties as minute brown specks. Through these lenticels air passes into and out of the apple; and water brought from the roots passes out as vapor. This process of water elimination is known as transpiration. Great quantities of water pass out in this way if conditions are favorable. Low humidity, high temperature, rapid movement of the wind, and intense light facilitate the process. If plenty of water is supplied by the roots, the process of transpiration removes the great bulk of it as vapor through the lenticels. If the supply of water is deficient, but at the same time conditions favor transpiration, then too much water is removed and wilting results.

"When the brown spots are examined with a compound microscope it is found that the affected cells are collapsed or perhaps broken, and hence the tissue sinks, forming a pit. Whether the cells are merely collapsed or are broken is a question yet undecided. The cell contents are lacking for the most part, only starch grains, which were not converted into sugar during the ripening process, remain. Their presence is regarded as proof that the injury to the cells occurred before the ripening of the fruit. The cells are brown and have every evidence of being dead. Their brown color and their lack of sap, and therefore their dry texture, give the described appearance to the whole diseased area. Careful search will show that the brown spots always arise in close connection with a branch of the sap-tube system.

"With these facts in mind the most important theories, which have been championed to explain the killing and drying out of the affected cells, may now be examined.

"(1) The injury is due to a rapid transpiration, or loss of water, from the cells, thus bringing about a concentration of the sap in these cells nearest the sap-tubes. This concentration of sap which is chiefly a concentration of the acids, results in the injury and death of the cells. The concentration of the cell-sap is therefore the immediate cause as outlined by this theory, and this concentration is increased by an insufficient water supply as well as by excessive loss of water. Those who cling to this theory believe that it accounts for the development of the disease both in storage and on the trees, and in the latter case for its appearance in wet as well as in dry weather. The following facts form the basis for this proposed explanation:

(a) Warm, dry weather favors transpiration.

(b) During such weather the soil is deplete in moisture and therefore cannot supply the cells with water sufficient to offset the rapid loss by transpiration.

(c) During the wet season the apple-fruit grows very rapidly and the net-work of sap-tubes is not able to keep pace with the growth of the pulp-cells and the proper balance is disturbed. The sap-tube system is then deficient and the cells suffer from a lack of water.

(d) The disease is favored by alternating wet and dry weather. Fluctuating temperature and humidity near the ripening period are highly favorable to bitter-pit, and conversely as long as the water is regularly supplied to the fruit, or at least as quickly as it is lost by transpiration, there is no pitting. Dry weather suddenly followed by heavy rainfall results in the development of stippen on the tree. And excessive transpiration, such as occurs in warm, dry weather, with a light crop of fruit is favorable to the disease. In other words, when transpiration is relatively greater than the water supply, stippen is produced. It is seen then that the weather conditions play a prominent role in causing this disease while the fruit is still on the tree. On the other hand, the methods of orchard management are contributing factors not to be overlooked. Heavy pruning is thought to favor the disease, for pruning affects transpiration. When the balance between the portions of a tree above ground and the roots

under-ground is disturbed, injury is likely to result. The application of manures not only supplies food but modifies transpiration. Green-manures conserve moisture in the soil. Manures of any kind invigorate the tree and enable it to use water from the soil in a more economical fashion. Cultivation conserves moisture and effects transpiration in a manner favorable to the tree. Irrigation must be taken into account. Regular and continuous growth is inimical to the development of bitter-pit. But if water is applied through irrigation in an injudicious fashion, conditions are made favorable to the disease. The disease also develops in storage. It has been found that fruit apparently clean when picked will show stippen after a considerable period in storage. Conditions in storage favorable to the disease are similar to those favoring it on the tree. Fluctuating temperature and humidity combined are evidently responsible for the production of bitter-pit. On the other hand, a uniform, low temperature and a dry air are unfavorable to the disease.

"(2) The second theory brought forward to explain the cause of stippen is that the injury is due to excessive transpiration of water from the fruit during warm days followed by reduced transpiration during the cool nights. A warm day results in rapid absorption of water by the roots and in its rapid loss by transpiration. The ensuing cool night checks the elimination process, but the soil remaining warm allows the roots to go on pumping water into the tissues of the fruit. This results in an internal pressure which finally bursts the distended cells. They soon die and turn brown, and give rise to the symptoms of stippen.

"(3) A third theory is based on the assumption that the disease is most destructive in dry seasons. The premature dryness of the soil results in a failure of sufficient mineral food to reach the fruit, in consequence of which certain groups of cells starve and perish.

"Many other causes have been assigned to stippen. Among the more prominent ones may be noted: local poisoning by spray material, poisons absorbed by the roots, bacteria, fungi, insects, mechanical injury, unfavorable grafting, degeneration from old age, and finally a peculiarity of the variety. A discussion of these various factors as causes of bitter-pit seems out of place in the present state of knowledge regarding the disease. None of them is known to be either direct or contributory causes of stippen.

"Control of bitter-pit..... It is very important that everything possible be done which will tend toward the production of good crops evenly distributed over the tree year after year; for herein lies a possible solution of the control of stippen. Most growers know that vegetative growth is unfavorable to fruitfulness. On the other hand, injudicious, heavy pruning to eliminate the vegetative growth also favors bitter-pit. It is believed that above all other orchard operations pruning has the most direct effect on the development of stippen. For it may regulate the amount, and to a certain extent the size, and it may determine the distribution of the fruit on the tree. The system adopted should be carefully planned, it should aim at the production of a regular crop of evenly distributed fruit, and should therefore be light..... Regulated sap-flow favors fruitfulness, and is also unfavorable to bitter-pit..... Trees with a light crop and unusually large-sized fruit are most subject to pit. Again, overcrowding favors the disease..... The water-elimination process in its

relation to bitter-pit has been emphasized. Although long years of experimentation are necessary to determine the effect, if green-manuring were practiced in non-irrigated orchards the disease would probably be reduced. Cover-crops are planted by many of the leading apple-growers. These crops aid in regulating the water-supply of the apple, and when plowed under will conserve the soil moisture. The question of drainage should not be overlooked. This disposes of excessive water during the days of heavy rainfall, and conserves moisture during drought-periods. The disease is worst in low, wet portions of an undrained orchard. Moderation should be practiced in the matter of irrigation. The heavy applicatior of water following a drought-period is favorable to stippen development. Excess should be avoided, and the aim should be to maintain uniformity of moisture conditions. Thorough orchard cultivation is advised. This operation affords proper aeration of the soil and consequent normal and efficient root action. It conserves moisture during periods of dry weather, and as a result transpiration is properly regulated."- Hesler, Lex R. and Herbert Hice Whetzel. Manual of Fruit Diseases. pp. 25-33.

The following quotation from Herbert (12) sets forth a new theory, the "crushed cell" theory, as opposed to the "bursting cell" theory originally proposed by McAlpine and mentioned in the above quotation.

The Crushed Cell Theory.

"The Bursting Cell Theory as summarized by McAlpine is as follows: When there is an extra rush of sap following on dry conditions, the rapidly swelling pulp cells at the external boundary burst the vascular network at localized spots and the sap pressure which is sufficient to rupture the enveloping network also bursts the thin-walled pulp cells at these particular spots and death of the cells ensues. Briefly, it may be stated that rapid alternations between dry and moist conditions, combined with fluctuating temperatures, during the growing stages of the fruit are the enciting causes of bitter pit.

"There are several points in this theory which are not supported by the following facts:

a. Sunken spots constitute the first external symptoms.
b. The skin is intact over the pit areas.
c. The vascular bundles frequently extend through a pit spot and supply normal tissue beyond.
d. Starch is present in the pit cells.
e. The tensile strength of cellulose is sufficient to eliminate the possibility of bursting.....

"It is generally accepted that the fluctuation in water supply is the factor primarily responsible for bitter pit. The point in question, however, is in what manner the diseased patches are produced. The rush of sap to the apple after a fall of rain following a dry spell causes a distension of the parenchymatous pulp cells. If the cells in any particular pit area were to distend to such an extent as to cause their bursting the result would be the local production of wart-like bodies before the bursting and subsequent death took place. As a matter of fact, a sinking of the tissue at the affected points constitutes the first external symptoms observed. In the case of Dunn's Seedling (Monroe's Favourite) the flow

of sap after a rain following a dry season is sufficient to cause the expected distension of the pulp cells to take place. The force of the distension is sufficient to cause a bursting of the skin. No bursting of the cells takes place within the apple; in fact, it is difficult to see how this could be possible because the expansion of adjacent cells would bring the tissue into a stage of static equilibrium and the only relief possible for the increased pressure would be either a bursting of the skin (as in the Dunn's seedling) or a crushing of some of the cells. There is not sufficient internal space in the apple for the expansion of cells to the bursting point. If cells are dissected out and placed in water the amount of water imbibed is not sufficient to cause bursting although under this condition they are freed from the natural obstacle of adjacent turgid cells which is found in the unruptured apple, and which helps to prevent their expansion. An osmotic pressure of 100 atmospheres when entirely borne by the cell may cause bursting but this is enormously beyond anything met with in the cells of the apple. In this case there is the further factor of the pressure from neighboring cells so that bursting of these internal cells is impossible.

"The vascular tissue may frequently be traced through a pit and found to be supplying healthy tissue beyond. This would not be the case if the vascular network were burst as the Bursting Cell Theory assumes.

"The presence of large quantities of starch in the dead cells is another point to be considered. Pitting takes place in the apple for the most part at the time when the starch is being converted into sugar. It is evident that when there is a sudden rush of sap into the apple, the cells which have had their starch converted into sugar will swell to a greater extent and more rapidly than those cells which are still supplied with starch. The rapid distension of the cells is resisted on the outside by the skin. Their force of expansion results in the crushing of those cells whose starch transformation is backward. This accounts for the presence of quantities of starch in the dead pit cells.

"It is suggested, though it remains to be proved, that immunity from bitter pit, which is shown in such varieties as Yates, may be due to the uniform transformation of the starch to sugar through the tissue. If such is the case there would be no small clusters of cells far enough behind in their starch transformation to be crushed by neighboring cells of a higher osmotic pressure."- Herbert, D. A. Bitter pit in apples: the crushed cell theory. Phytopathology 12: 489-491. October, 1922.

References.

(1) Hesler and Whetzel. Manual of Fruit Diseases. 1917. pp. 23-34.

(2) McAlpine, D. Bitter Pit Investigations.

(3) Herbert, D. A. Bitter pit in apples: the crushed cell theory. Phytopathology 12: 489-491. Oct., 1922.

(4) Brooks, Chas. and D. F. Fisher. Irrigation experiments on apple spot diseases. Jour. Agr. Res. 12: 109. Jan. 21, 1918.

CORK AND DROUTH SPOT.

Cause.- disturbed water relations.

1. Laboratory Study.

a. Examine specimens and determine the nature of the injury as nearly as you can. Examine sections of diseased and healthy tissue under the microscope. Draw and describe gross symptoms and features revealed by the microscope.

2. Text discussion.

Mix (11) describes these troubles as follows:

"History and Distribution.

"Cork has apparently been known locally for at least fifty years. There is, however, little mention of it in literature, at least in a way which would allow it to be distinguished from the true fruit-pit or stippen. Craig (8) notes Fameuse as susceptible to stippen or "dry rot" as he terms it, and from the description by Selby (21) of specimens of Fameuse which he had received from Craig it is apparent that here was a case of typical cork. Selby (22), in a discussion of "brown spot" or "dry rot" of Baldwin, mentions internal spotting as occurring without external lesions in the varieties Fameuse and Northern Spy. Cobb (7) describes an "obscure disease" of the apple occurring in New South Wales. McAlpine (13) states that he visited this locality in the season of 1912 and found the same disease in various orchards. It was confined to the variety Pomme de Neige (Fameuse). The same author (14) again mentions this disease, stating that it is a type of bitter-pit occurring when the apples are very small, the irregular knotty appearance being due to subsequent growth. His figures, as well as those of Cobb, show that the "obscure disease" is identical with the cork of Fameuse as found in the Champlain valley.

"Drouth spot, so far as is known to the writer, had not been mentioned in literature prior to his first observations in Clinton county. Specimens were submitted in 1913 and again in 1914 to Dr. Charles Brooks, of the Bureau of Plant Industry, and in 1914 Dr. Brooks visited Clinton county and went over the situation with the writer. He stated that he had never observed the trouble, although he had seen somewhat similar troubles in the irrigated section of the Pacific Northwest. Dr. W. J. Morse, of the Maine Agricultural Experiment Station, who saw specimens of the disease in July, 1914, expressed the opinion that he had seen apples similarly affected in Maine. Recently, Reed and Crabill (20) have described a "skin crack" of the York Imperial in Virginia. The writer is convinced that this was a case of drouth spot that was not observed until a late stage ("early in September"). It is mentioned as prevalent in 1914 and as having been noticed previously only in very dry seasons. Brooks and Fisher (3) under the heading 'corky pit or drouth spot', describe an apple disease occurring in the Northwest which, if not identical with the drouth spot, is at least very similar to it. They also state: 'There is another form of corky pit that occurs on shallow soils in some sections of the East and West. It is common where there is a hardpan at a slight depth and also where there are peculiar soils outcropping....'

"Allen (1) in a report on the study of the root condition of apple trees in the Hood river valley, makes the following statement: 'Some trees were found to be so seriously devitalized by drouth and starvation that the fruit was not only badly pitted but hollow in places. On such trees the foliage was very small; a large portion of it had fallen; and the ends of many twigs had begun to die back.' The illustration of a pitted apple accompanying this article is more suggestive of certain severe types of drouth spot than it is of true fruit-pit or stippen.

Economic Importance.

"Whether it drops from the tree, as in drouth spot occurring early, or persists and becomes deformed, as in cork and late appearing drouth spots, an affected fruit becomes a total loss. Cork usually takes the whole crop of affected trees or of individual branches year after year. In some cases, in favorable years, it has been known to affect the entire Fameuse crop of some orchards. Drouth spot may involve only a few fruits on an affected tree, or may, in some cases, ruin its entire crop. In one orchard, in 1914, it was estimated that the loss from drouth spot was two hundred barrels. Eleven hundred were actually harvested. This, however, is extreme.

"Cork - symptoms.

"Cork is most commonly observed when the apple is in a state anywhere from half grown to nearly mature. It may be briefly characterized as internal browning.....without external pits, but with the surface of the apple thrown into a series of elevations and depressions. A large number of brown corky areas occur throughout the flesh, following closely the course of the vascular bundles. In no case do these extend outward as far as the skin, consequently there are none of the external brown pits characteristic of true fruit-pit or stippen. A further difference from the usual type of fruit-pit is that the spots are not more abundant in the peripheral zone, but are scattered throughout the flesh of the fruit. There is no bitter taste connected with this disease in Fameuse apples.

"In the earliest observed stage of the disease, when the fruits are about two centimeters in transverse diameter, there is no external evidence of an abnormal condition. The shape of the fruit and the color and appearance of the skin are normal. Upon cutting open the fruits, comparatively large, brown, corky areas are seen at various places in the flesh. These are often near the periphery directly opposite one of the main vasculars, or they may be, in some cases, arranged irregularly within the core lines around the core. Sometimes, in the earliest stage of the disease, the internal brown spots are surrounded by a green water-soaked area suggestive of the well known water-core disease. This area may even show through the skin of the fruit as a darker green water-soaked spot. This appearance is not, however, characteristic of the disease and is not found in later stages.

"Drouth spot- symptoms.

"The lesions of drouth spot may be divided into two general types,--superficial and deep seated. They also vary somewhat according to the age and size of the fruit when affected, and the age of the lesion.

"Superficial lesion.- In this case an early stage of the disease is manifested by an irregular, light-brown area in the skin. When the fruits affected are large, two or three centimeters in transverse diameter, the surface of the fruit is usually smooth and regular, there is no shrinkage or sinking in, nor any abnormality in the flesh beneath. On large fruits, also, the lesion occurs often on the cheek of the fruit and is not more common toward the calyx end. When the fruits are smaller, and sometimes on larger fruits, the spots show an irregular, wavy pattern, formed by a series of wrinkles or corrugations the elevations and depressions of which are, however, very small, and it is rare that there is any abnormality in the flesh of the apple. It is as though the browned areas marked the course of the fine network of vascular endings situated just beneath the skin of the fruit. (See McAlpine (5) and Brooks (10). This idea receives confirmation from a study of cross sections through such lesions placed under the microscope. On young fruits, also, the disease occurs normally toward the calyx end of the fruit, often as a broad collar of diseased tissue just below the base of the calyx lobes.

"Deep-seated lesion.- This type of lesion is characterized by the presence of brown, corky areas in the flesh of the apple and by a sinking in of portions of the epidermis. On young fruits, from one to two or two and one-half centimeters in transverse diameter, the disease appears as a large brownish area in the skin of the fruit, usually near the blossom end, which is irregularly sunken and wrinkled, indicating shrinkage of the tissues beneath. Cross sections show brown areas in the flesh near the periphery. These are opposite the main vasculars, and often in the center of one of them there is a large cavity, the apex of which reaches one of these vessels. (Occasionally, apples are found in which there is one of these corky areas or cavities opposite each of the ten main vasculars.) These internal spots are often connected by a narrow brown streak running close to the periphery of the apple. Sometimes these streaks do not connect, but extend only a short distance in either direction from the central spot. The shrinkage of the skin over a considerable area, and the presence of these brown corky spots and streaks in the periphery suggest the type of fruit-pit described by McAlpine (5) as 'confluent bitter pit' or 'crinkle'.

Control

"The only control method which offers promise of results is one looking to conservation of soil moisture, and an even distribution of the moisture supply throughout the season. Clean cultivation is not sufficient. From certain suggestions of benefit in the Champlain valley, and from results secured in the Pacific Northwest, clean cultivation followed by a leguminous cover crop, or the planting of a leguminous crop such as alfalfa in the orchard, seem methods which should be tried....."- Mix, A. J. Cork, Drouth spot and related diseases of the apple. New York Agri. Expr. Sta. Bul. No. 426. October, 1916.

References.

(1) Craig, J. A dry rot of apples. Canada Expt. Farms. Rpts. 1896: 171-172. 1897.

(2) Selby, A. D. Some diseases of orchard and garden. Ohio Sta. Bul. 79: 135. 1897.

(3) _____ A brief handbook of the diseases of cultivated plants in Ohio. Ohio Sta. Bul. 214: 369. 1910.

(4) Cobb, N. A. An obscure disease of the apple. Agr. Gaz. N. S. Wales 3: 284. 1892.

(5) McAlpine, D. Bitter pit investigation. The past history and present position of the bitter pit question. First progress report. 197 pp. 1911-12.

(6) _____ Bitter pit investigation. The cause of bitter pit: its contributory factors together with an investigation of susceptibility and immunity in apple varieties. Second progress report. 224 pp. 1912-13.

(7) Reed, H. S. and Crabill, C. H. Notes on plant diseases in Virginia observed in 1913 and 1914. Skin crack of the York Imperial. Va. Sta. Tech. Bul. 2: 47-49. 1915.

(8) Brooks, C. and Fisher, D. F. Spot disease of the apple causing much general confusion. Better Fruit 10: No. 8: 13-15.

(9) Allen, R. W. Condition of root system of apple trees in the Hood River district. Ore. Sta. Rpt. Hood River Branch Sta. 1914-15: 20-24. 1915.

(10) Brooks, C. The fruit spot of apples. Bul. Torrey Bot. Club. 35: 423-456. 1908.

(11) Mix, A. J. Cork, drouth spot and related diseases of the apple. N.Y. (Geneva) Exp. Sta. Bul. 426. 1916.

(12) Barss, H. P. Physiological disorders of developing fruits. Ore. Exp. Sta. Crop Pest and Hort. Rep't. 1915-20: 159-166. 1921.

JONATHAN SPOT.

Cause not definitely determined.

References.

(1) Hesler and Whetzel. Manual of Fruit Diseases. 1917. pp. 137-140.

(2) Scott, W. M. and Roberts, J. W. The Jonathan fruit-spot. U. S. D. A. Bureau Plant Industry Cir. 112: 11-16. 1913.

(3) Jackson, H. S. and J. R. Winston. Jonathan fruit spot. Oreg Exp. Sta. (Hood River Branch) Rept. 1913-1914: 20-21. 1915.

(4) Norton, J. B. S. Jonathan fruit spot. Phytopath. 3: 99-100.

(5) Cook, M. T. and G. W. Martin. Jonathan Spot rot. N. J. Exp. Sta. Rept. 1914: 500-503. 1915.

(6) _____ The Jonathan spot rot. Phytopathology 3: 119-120. 1913.

(7) _____ The Jonathan spot rot. Phytopathology 4: 102-105. 1914.

WATER CORE OF APPLE

Caused by abnormal water relations.

References.

(1) Norton, J. B. S. Water core of apple. Phytopathology 1: 126. 1911.

(2) O'Gara, P. J. Studies on the water core of apple. Phytopathology 3: 121. 1913.

(3) Clinton, G. P. Report of the Botanist. Water Core. Conn. Exp. Sta. Rept. 1913: 8-9. 1914.

(4) Hesler & Whetzel. Manual of Fruit diseases. 1917. pp. 120-124.

APPLE SCALD

Caused in part by poor ventilation.

Brooks, Cooley and Fisher (3) describe this trouble as follows:

"Scald is a transportation and storage disease that is produced by the gases given off by the apples themselves. Under the prevailing methods of handling fruit it causes greater losses than all other storage diseases combined. It is particularly serious in the York Imperial, Grimes, Arkansas (Mammoth Black Twig), Rome Beauty, Rhode Island Greening, Stayman Winesap, Wagener, and Baldwin varieties, and it occurs at times on almost every variety of apple.

"Scald can be distinguished from all other apple diseases by its preference for the greener side of the apple. In mild cases of the disease the apple is merely tinted with brown, but in more severe cases the entire skin layer is killed and sometimes broken down to the extent that it will slough off readily from the flesh. In some instances the flesh becomes dead and brown to a depth of half an inch, and the disease takes on the appearance of an apple rot; but true rot usually spreads down into the flesh in more or less conical shape, while scald is usually diffuse, spreading over a large area without having much depth. An apple that has had its skin killed by scald becomes the ready prey of the various rot organisms, and they soon finish the work of destruction that the scald has begun.

"It is a rather generally accepted idea that scald is due to the warming of the fruit after it has been removed from cold storage. This idea comes from an erroneous interpretation of very familiar facts, and this misconception in regard to the nature of scald has often resulted in throwing the responsibility for its

occurrence upon the wrong party. It is true that apples usually show but little scald while held continuously in cold storage and also true that fruit apparently free from disease may become badly scalded after exposure to warm air for a few days, but the apples were already potentially scalded while still in storage and merely required the open air and the higher temperatures to allow their death processes to be completed. The real cause of scald is to be found in the accumulation of certain gases (esters) given off by the apples themselves, and prevention can be had by removing these gases by ventilation or by absorbing them with oils. There are a great many fats and oils that will accomplish this result, but the mineral oils sold under various trade names and refined so as to be practically or entirely free from odor and taste, have been found the most satisfactory. They are among the cheapest oils, they do not become rancid, and they are already extensively used on paper and otherwise in connection with food products. The most convenient method of applying the oil is by means of oiled wrappers. The development of scald is much more rapid at high than at low temperatures. Most varieties of apples are scalded as badly at 60° F. in one month as at 40° F. in three months.

"The responsibility for scald does not always lie in the same quarter. Its occurrence is influenced by orchard, packing-house, and transportation as well as storage conditions. Large apples and those that have been forced late in the season by heavy irrigation or rains have an increased susceptibility to scald. Apples that have been scarred and russeted by powdery mildew are rendered more susceptible, while those from trees that have been badly affected with cedar rust are very resistant to scald. Fruit that is picked green is much more susceptible than that which is left on the trees till well matured. The holding of apples in closed packing sheds or in unrefrigerated cars may be responsible for the development of scald later in storage or upon removal from storage. Placing large quantities of warm fruit in a particular storage room instead of distributing it in several rooms is likely to result in delayed cooling and in heavy losses from scald.

"Apples scald far less in boxes, crates, or ventilated barrels (barrels with 15 holes three-fourths by 4 inches cut in the staves) than in the usual commercial barrels, especially if the storage room is allowed considerable change of air. They scald far less when located near the door of the storage room than when in the back corners away from the aisles. False floors, wall spaces, openings between the stacks, the operation of fans, and the throwing open of storage windows when the outside temperature will allow, all contribute to scald prevention.

"Scald can be entirely prevented by storing apples in oiled (not waxed) wrappers. This treatment has been found completely successful even when susceptible varieties have been placed in unventilated storage and held far beyond their usual storage season. A good quality of oiled wrapper can be prepared by hand-oiling the usual commercial wrapper, but the cost of the labor is too great. The oiled paper now on the market has been prepared for other purposes and is too heavy for convenient use on apples. Lighter weight oiled papers suitable for use as apple wrappers are now in process of preparation."

References.

(1) Brooks, Chas., J. S. Cooley and D. F. Fisher. Apple-scald Jour. Agr. Res. 16: 195. Feb. 24, 1919.

(2) _____ Nature and Control of apple-scald. Jour. Agr. Res. 18: 211. Nov. 15, 1919.

(3) _____ Diseases of apples in storage. U. S. D. A. Farmers' Bull. 1160. pp. 21-23. 1921.

(4) Brooks, Chas. and J. S. Cooley. Effect of temperature, aeration and humidity on Jonathan spot and scald of apples in storage. Jour. Agr. Res. 11: 287-318. 1917.

BLOSSOM-END ROT, OR POINT ROT OF TOMATO.

Probably caused by unbalanced water relations.

Jackson gives the following brief description of this disease:

"This disease is quite generally distributed throughout the country. It seldom causes extensive loss, but from the fact that it is more serious on the early crop, the financial loss becomes a factor of importance.

"This rot may occur at various stages of development of the fruit, generally before it has reached full size, and, as has been stated, early in the season. It makes its appearance at the blossom end in the form of sunken brown spots which gradually enlarge. Such spots are dry and hard. Many times molds or bacteria gain entrance at this point when the fruit is imperfect and cause decay. Such troubles should not be confused with the one under consideration although they frequently follow it.

"The true cause of point rot does not seem to be thoroughly understood.

"It is known that the trouble is more serious in dry weather on light soils where the supply of water is insufficient. This can be corrected either by irrigation or by adding organic matter so that the water-holding power is increased. Surface tillage should also be practiced. Experimental work has shown that subirrigation is better than surface watering and that where plants are shaded so that transpiration is reduced the amount of rot is considerably lessened."- Jackson, H. S. Tomato diseases: Blossom-end rot or point rot. Ore. Exp. Sta. Biennial Crop Pest and Horticultural Report. 1911-1912. p. 290. 1913.

INTERNAL BROWN SPOT OF POTATO.

Probably caused by unbalanced water supply.

McKay gives the following brief description of this trouble.

"The development of irregular brown spots in scattered and irregular places in the interior of potato tubers is known as internal brown spot. These brown spots are free from bacteria or fungi and represent merely groups of cells which have died from some cause or other. This disease is generally believed to be due to lack of water at some period during the growth of the plants, perhaps as the result of poor soil or merely insufficient soil moisture. The trouble is not transmissible in the tubers. Affected potatoes are, however, generally avoided for seed on account of the chance for predisposition of the affected potatoes to this disease."- McKay, M. B. Potato diseases in Oregon and their control. Ore. Exp. Sta. Cir. 24. pp. 51-52. 1922.

BLACKHEART OF POTATO.

Caused by too high temperatures and poor ventilation.

McKay briefly describes blackheart as follows:

"The disorder of potato tubers known as blackheart is distinguished by the occurrence in the center of the tuber of an irregular area of black, quite moist, and flabby tissue. Occasionally the discoloration may occur as a black ring near the center inclosing a gray or darkened area. In advanced stages, the tubers show hollow, black-bordered cavities in the center. The discoloration progresses outward and whenever it reaches the outside, as occasionally happens, a rapid decay sets in.

"Blackheart is produced when potatoes are heated to 100° to 110° F. for a few hours or stored at lower temperatures in piles deeper than six feet without ventilation from the sides or bottom. This type of trouble is most frequently encountered in shipments of potatoes which have been overheated in transit, in bins where the ventilation is unusually poor, and in seed potatoes that are taken out of storage and exposed to too high temperatures in the sun for too long a period while they are being prepared for planting."- McKay, M. B. Potato diseases in Oregon and their control. Ore. Exp. Sta. Cir. 24. pp. 51-52. 1922.

References.

(1) Stewart, F. C. and A. J. Mix. Blackheart and the aeration of potatoes in storage. N. Y. (Geneva) Exp. Sta. Bul. 436. 1917.

(2) Bartholomew, E. T. Blackheart of potatoes. Phytopathology 3: 180-182. 1913.

FREEZING INJURY.

There are many types of freezing or frost injury ranging all the way from that caused by untimely late spring frost to blossoms, new shoots and young fruits to the killing of trees by severe freezes in winter and the damage done to fruits and vegetables when allowed to freeze in storage. The following list of references cover practically the whole range of injuries caused by low temperatures, as the titles indicate.

References.

(1) Hesler and Whetzel. Manual of Fruit Diseases. 1917. pp. 35-44.

(2) Mix, A. J. Sun-scald of fruit trees: a type of winter injury. Cornell Univ. Exp. Sta. Bul. 382. 1916.

(3) Bradford, F. C. and H. A. Cardinell. Observations on winter injury. Mo. Exp. Sta. Bul. 56. 1922.

(4) Chandler, W. H. The killing of plant tissue by low temperature. Mo. Exp. Sta. Res. Bul. 8: 143. 1913.

(5) O'Gara, P. J. The protection of orchards in the Pacific Northwest from spring frosts by means of fires and smudges. U. S. D. A. Farmers' Bul. 401: 5-24. 1910.

(6) Grossenbacher, J. G. Crown rot of fruit trees: field studies. N. Y. (Geneva) Exp. Sta. Tech. Bul. 23: 3-59. 1912.

(7) Whipple, O. B. Winter injury to the fruit buds of the apple and the pear. Montana Exp. Sta. Bul. 91: 35-45. 1912.

(8) Jones, L. R., M. Miller and E. Bailey. Frost necrosis of potato tubers. Wis. Exp. Sta. Res. Bull. 46. 1919.

(9) Wright, R. C. and George F. Taylor. Freezing injury to potatoes when undercooled. U. S. D. A. Bul. 916. 1921.

(10) _____ The freezing temperatures of some fruits, vegetables and cut flowers. U. S. D. A. Bul. 1133. 1923.

(11) Selby, A. D. Fall and early winter injuries to orchard trees and shrubbery by freezing. Ohio. Exp. Sta. Bul. 192. 1908.

CHAPTER XXVI.

Diseases of unknown cause.

In ancient and medieval times all plant diseases were more or less mysterious. During the modern and present eras the nature and cause of a vast number of diseases have been worked out more or less completely. Even so, there always have been and probably always will be coming to the attention of plant pathologists from time to time diseases of obscure nature. Eventually most if not all of these puzzling troubles will yield up their secrets to the investigator. However, as long as there are such diseases, any system of classification based upon cause must provide a pigeon hole, a sort of phytopathological catch-all, where these diseases can be temporarily classified until sufficient knowledge is available for their proper classification.

There is neither time nor space to go into detail here. We simply call attention to the fact that practically every issue of Botanical Abstracts has listed a group of articles under the heading "Diseases of unknown cause". Some of these diseases will undoubtedly be transferred in a short time to a group of known cause while others may have to remain in the obscure group indefinitely.

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